Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

AVMA Convention—Philadelphia, August 18-21, 1958

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Correspondence

March 4, 1958

Dear Sir:

I am enclosing two articles for your consideration.

The first is an original poem which has been published in the *Wisconsin Veterinary Science News* (Summer, 1957). However, I thought perhaps its appeal was universal enough to be of possible interest for publication in the JOURNAL.

My Little "Verry-Narry"

I have a little boy at home,
His eyes are black and merry,
He always tells the kids around,
"My Dad's a 'Verry-Narry.' "

"He gives the cows their shots and things,
And checks a dog or cat,
He Doctors up the squeal pigs,
And does all things like that."

"When I grow big and strong like him,
Chance I'll make some calls,
I'll treat the cows, and pigs, and stuff,
In my boots and 'underhalls.' "

We often laugh at what they say,
And wonder what they mean,
But you'd surely miss the trust they have,
Or how big to them you seem.

So when next your cares are extra large,
And the skies are gray and dark,
Hark to the words of that little one,
Whose trust rides in your heart.

He looks at you and speaks these words,
With eyes so black and merry,
"When I be big and strong like Pop,
I'll be a 'Verry-Narry.' "

—J. H. ROSENBERGER, D.V.M.
Clinton, Wisconsin.

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Philadelphia Preconvention Conference

Past Preconvention Conferences, providing an opportunity for members of the profession to hear and participate in discussions of pressing problems, have been well received. The last two have dealt with veterinary relations with the press, radio, and television and internal and external public relations within constituent associations.

This year, ten speakers will discuss the organization of local and state associations, their interrelationships, and the reflections of this relationship in the public relations efforts of the profession. Strong associations will report their sources of strength, and examples of successful public relations activities will be presented in a how-to-do-it fashion. Following, is the complete program:

Preconvention Conference on Veterinary Associations and Public Relations

Chairman—H. E. Kingman, Jr., Director, Interprofessional Relations, AVMA

Sunday, August 17

9:30 a.m. Coffee and Donuts (Compliments of the AVMA)

10:00 a.m. Strong Local Associations Are the Building Blocks of Public Relations
W. W. Armistead, President, AVMA

10:30 a.m. A Strong Local Association in Action
Bruce Hostrawser, South Bend, Ind.; President, Michiana V.M.A.

11:00 a.m. The Women's Auxiliary Role in a Strong Local Association
(Speaker to be Announced)

11:30 a.m. An Example of Effective Local Public Relations—An Emergency Phone Service
C. E. De Camp, Scarsdale, N.Y.; Chairman, Committees on Public Relations of the AVMA and the New York City V.M.A.

Discussion

12:30 p.m. Luncheon (Speaker to be Selected)

2:00 p.m. Organization of an Urban Local Association
A. G. Madden, Cincinnati, Ohio; Chairman, Public Relations Committee, Cincinnati V.M.A.

2:30 p.m. Organization of a Rural Local Association
M. M. Leonard, Asheville, N. Car.; President, Western North Carolina V.M.A.

3:00 p.m. Building a Strong State on Local Associations
Bill Harris, Puyallup, Wash.; President-Elect, Washington State V.M.A.

3:30 p.m. Local Veterinary Association Profile (Survey)
H. E. Kingman, Jr.

4:00 p.m. Reflections of PR at National, State, and Local Levels
B. M. Forster, AVMA Director Public Information

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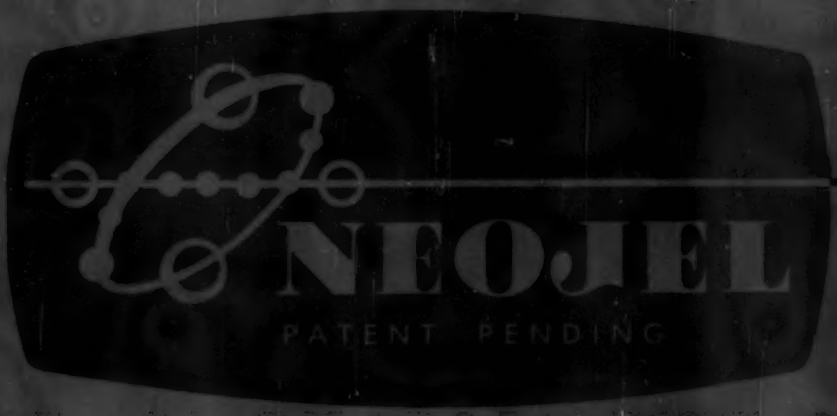

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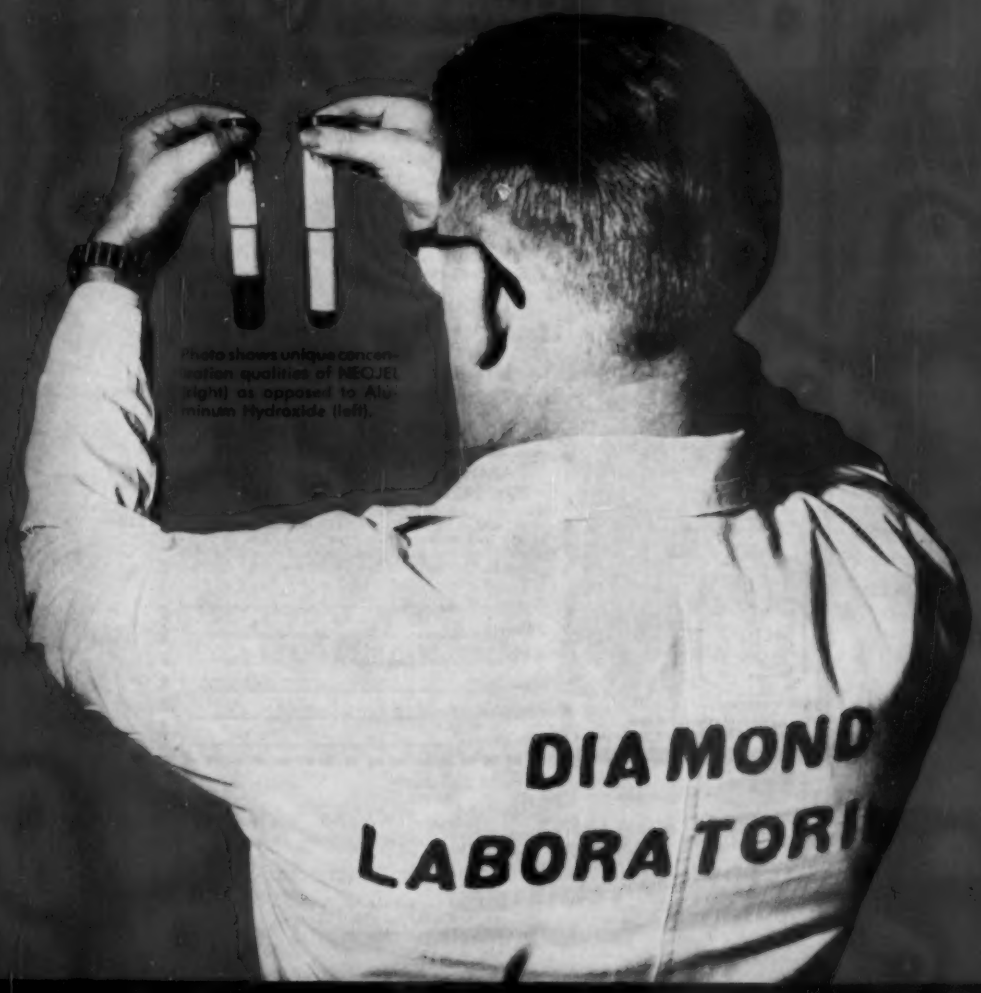

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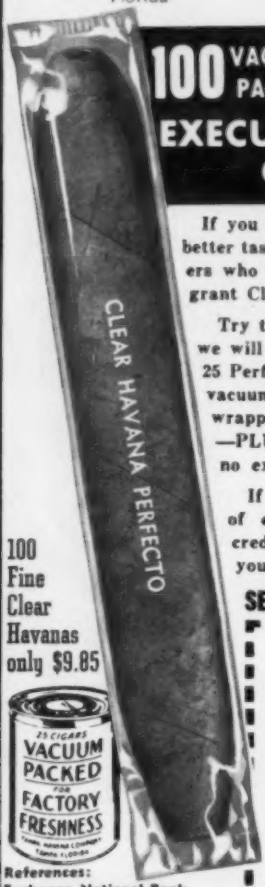

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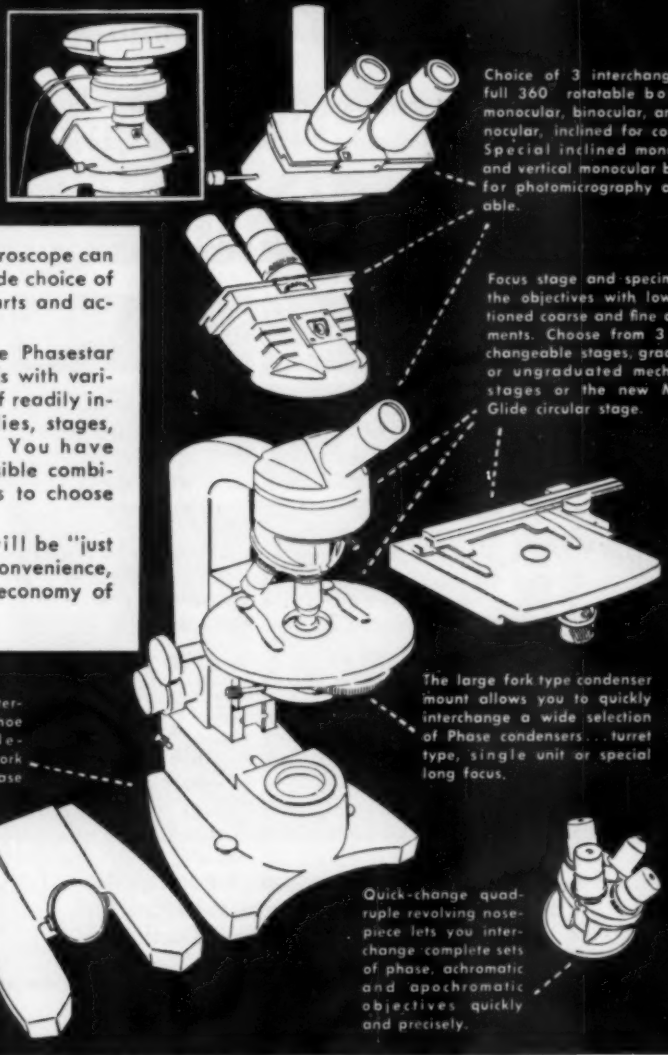

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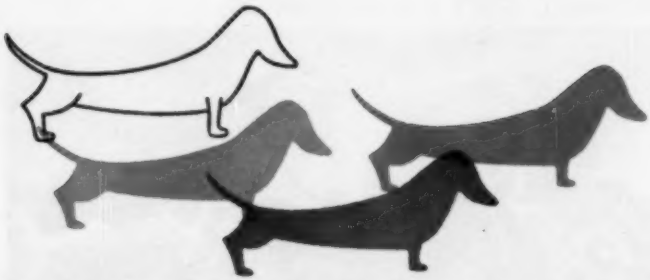

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1. Rachman, M., and Frucht, T. R.: Vet. Med. *49*:341, 1954.

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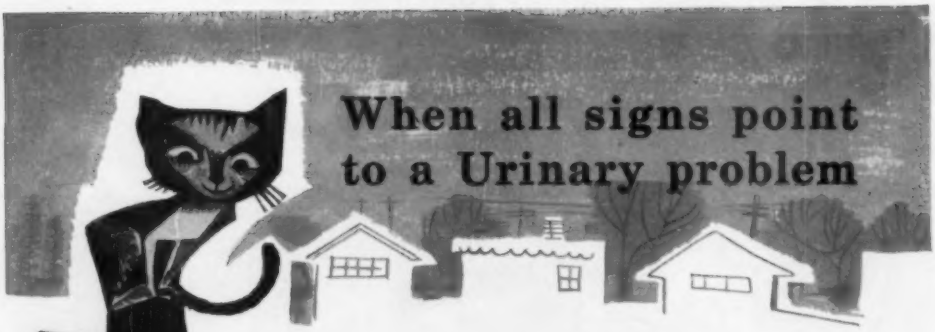

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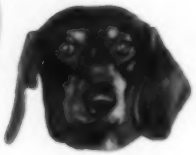

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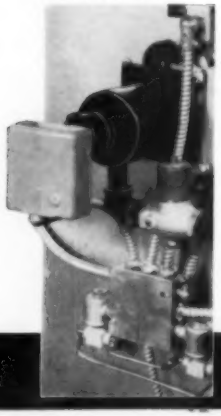

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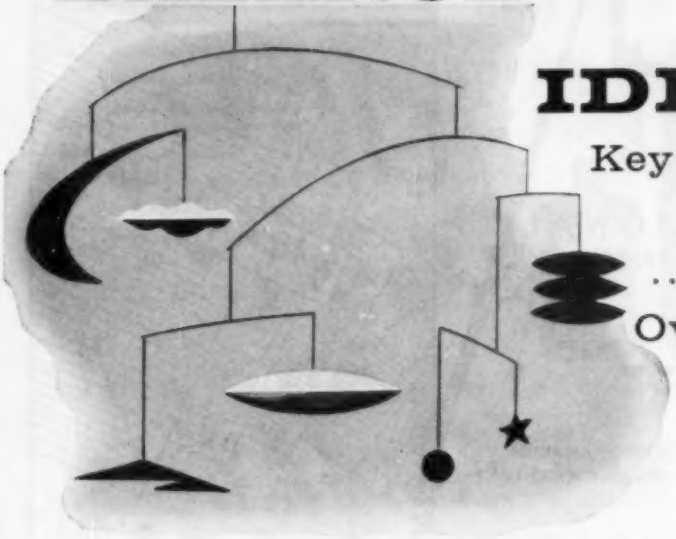

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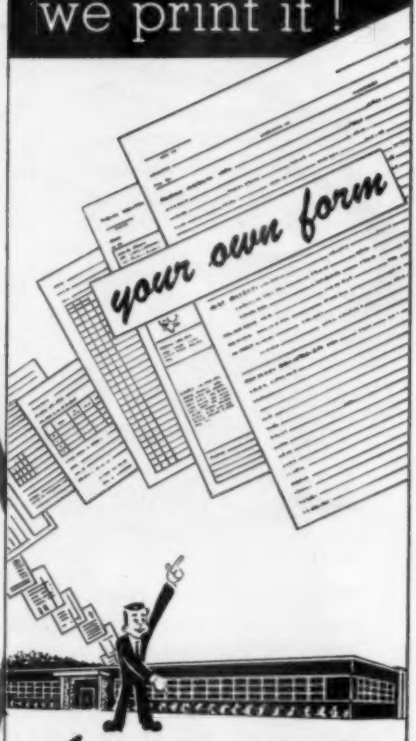

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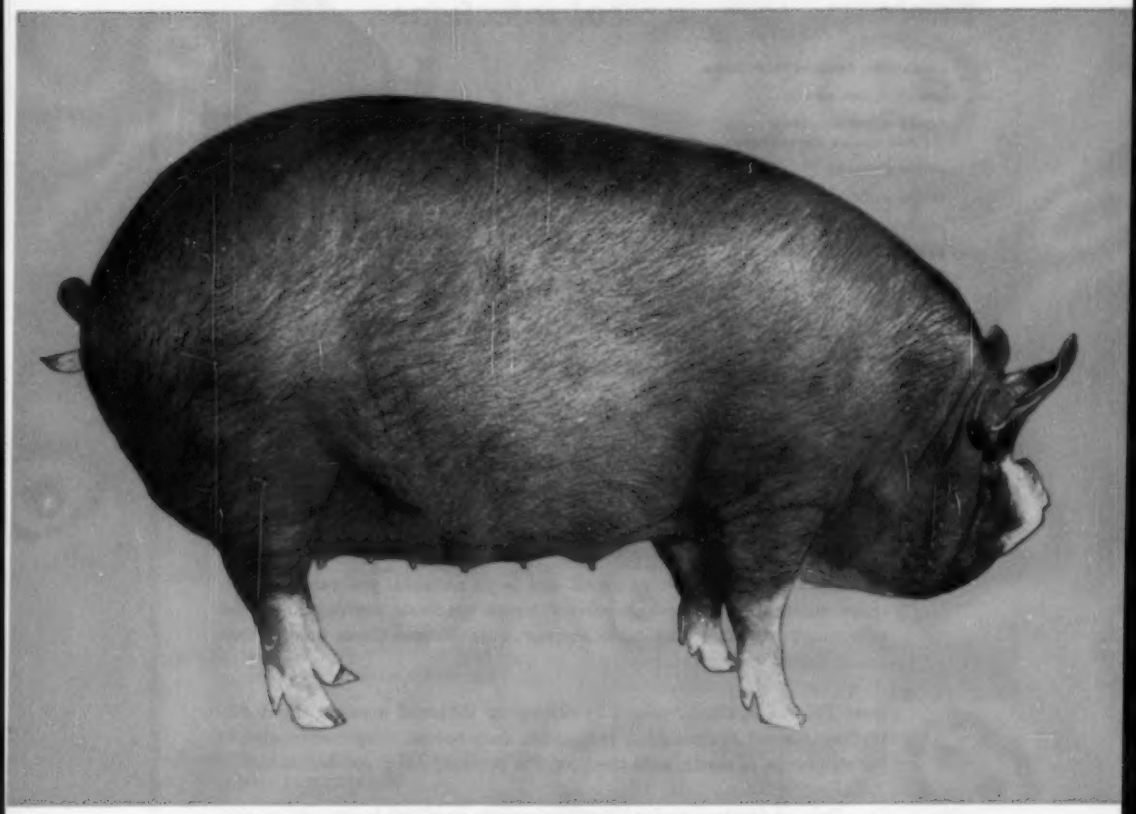

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The Effect of Stomach Contents and the Soil on the Viability of *Erysipelothrix Rhusiopathiae**

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Guelph, Ontario

THE SIGNIFICANCE of the carrier pig in the spread of *Erysipelothrix rhusiopathiae* is not clear. Some[7] have believed that the healthy pig is not a dangerous spreader. Others[6] believed that a certain number of carriers have probably, at one time, been exposed and resisted infection, whereas other swine harbored the organism without being afflicted.

Numerous investigators[2,3,5,10] have reported the presence of this organism in the tonsillar area of healthy pigs. The presence of *Ery. rhusiopathiae* in the tonsil may be the result of recovery from an attack of swine erysipelas,[12] and the apparently healthy carrier may retain the infection long enough to accomplish dissemination to new areas and new herds.[3]

For the carrier of *Ery. rhusiopathiae* in the tonsil to be important in the spread of erysipelas, it is probable that the organism must pass through the acid gastric juices of the stomach to the intestine, where it is able to reproduce in a favorable alkaline environment. By defecation, these carriers could infect the soil. A survey of the literature has not revealed any information concerning the effect of stomach contents of swine on the viability of *Ery. rhusiopathiae.*

It was reported in 1942[14] that sandy soils rich in lime and humus are especially favorable to the organism. In soil with an alkaline reaction, the viability of *Ery. rhusiopathiae* had not been reduced after 90 days, whereas acid soils destroyed the organisms within a few days.

That *Ery. rhusiopathiae* can live in certain soils has been reported by other workers. It was also reported[13] that earth obtained from regions where the disease was unknown did not lend itself to cultivation of the organism.

Although infection of the soil by the carrier pig may be important in the epizootiology of swine erysipelas, of equal significance is the length of time the organism remains viable in the soil. The present experiments were designed to observe the effect of stomach contents on the viability of the organism, and its persistence in soil from an area in which swine erysipelas had occurred.

MATERIALS AND METHODS

Stomach contents were collected, at an abattoir, from normal swine within a few minutes after slaughter, and by aspiration. The pH of samples was determined within a half hour after collection.

The strains of *Ery. rhusiopathiae* were those designated as "C" strains, which had been isolated by culture of the blood from swine in which the disease had been reproduced experimentally by means of dermal scarification. All strains were in the smooth phase when used. The strains were mixed in trypticase soy broth* and incubated for 24 hours at 37 C. It was estimated by use of McFarland's nephelometer that each milliliter of the incubated broth contained between 900 million and 1 billion organisms.

Three milliliters of broth culture was added to 15 Gm. of stomach contents from each pig and incubated at 37 C. for the following periods: 5, 15, 30, 45 minutes, and 1, 2, 3, 4, and 24 hours. The pH was determined electrometrically at the end of each time interval, and several loopfuls of the stomach contents were streaked on plates of trypticase soy agar containing 5 per cent, by volume, of sheep's blood.

From the Department of Research, Ontario Veterinary College, Guelph.

*Taken from thesis submitted to the University of Minnesota in partial fulfillment for the Ph.D. degree.

*Baltimore Biological Laboratories, Baltimore, Md.

The soil was sampled in the following areas at the Ontario Agricultural College: (1) boar pen, (2) main sow pen, (3) outer sow pen, and (4) a nearby farm belonging to the O.A.C. Swine erysipelas had occurred in each of these areas. The samples were collected in sterile quart jars or large clay pots. The pH of each soil sample was determined and then each sample was cultured on Packer's medium[9] for presence of *Ery. rhusiopathiae*.

The first study consisted of placing 15 Gm. of the soil in a sterile, screw-cap glass tube. This soil was autoclaved at 15 lb. pressure for 30 minutes and then dried in an oven for four days. The pH of the autoclaved samples was slightly higher than the original samples. Similar portions of soil were prepared without being sterilized.

Tubes of soil were inoculated with 1 ml. and 3 ml. of the broth culture, and incubated at room temperature.

Initially, the soil was cultured every four hours for 12 hours and then daily until cultures were negative. When a soil sample became negative, it was then diluted by mixing 1 part soil to 10 parts saline solution and further diluted to 10^{-4}. Packer's medium was inoculated with 4 to 5 generous loopfuls of these four dilutions. These plates were incubated for 48 hours and then examined under a dissecting microscope for evidence of the typical bluish granular convex colonies of *Ery. rhusiopathiae*. Suspicious colonies were spread on blood agar plates and stabbed into gelatin.

After the completion of these studies, the farm area soil, which had been the best for maintenance of the organism, was resampled. The soil samples were placed in three clay pots, two of which (pots 1 and 2) had a diameter of 8 inches and a depth of 8 inches. The soil in pot 1 was sterilized in the manner previously described. Pot 3 was 12 inches in diameter and 7 inches deep.

A hollow glass tube, 1 inch in diameter, was inserted into the soil of each pot to a depth of 4 inches; the soil was then removed from the lumen of this tube and tube was corked. Soil was sampled from the top of each pot, and also from the bottom of the glass tube in summer and autumn, but only from the surface of the pots in the winter. The pots of soil were maintained on the roof of a building exposed to the weather.

In the summer period, 500 ml. of broth culture was inoculated into the soil in pots 1 and 2, and 1,000 ml. of culture into pot 3. The soil used in the summer test was discarded and, for the remainder of the study, soil was obtained from the farm area previously sampled. In the autumn, a similar quantity of broth culture was used as in the summer except that it was divided into two equal amounts, inoculated at a two-day interval.

The soil in the pots was next inoculated after it had been negative for *Ery. rhusiopathiae* for four months (winter-spring test). At this time, the soil was inoculated six times, at two-day intervals, with 200 ml. of trypticase soy broth culture. When this soil in the pots became negative on culture for *Ery. rhusiopathiae*, 3 swine, 4 months of age, were drenched twice, at intervals of two days, with 500 Gm. of an 0.85 per cent saline suspension of the soil. In three weeks 2 of the 3 animals were destroyed and examined bacteriologically and pathologically.

The remaining pig was placed in contact with 3 normal litter mates and kept in the same pen for 14 days prior to preparation for the dermal scratch test.

Strains of *Ery. rhusiopathiae* isolated from the pots of soil at varying intervals after inoculation were rubbed into four scratches on 3 of the 4 swine; 1 was scratched but not inoculated. After the other 3 had been slaughtered, this animal was tested by the scratch method using strains of *Ery. rhusiopathiae* received from the U.S.D.A. (422-1,S192,NF4,HC595).

TABLE I—The Viability of Erysipelothrix Rhusiopathiae in the Stomach Contents of Swine

pH of stomach contents	Incubation									No. of tests
	5 Min.	15 Min.	30 Min.	45 Min.	1 Hr.	2 Hr.	3 Hr.	4 Hr.	24 Hr.	
1.1	+*	+	+	−*	−	−	−	−	−	1
2.0	+	+	+	−	−	−	−	−	−	1
2.8	+	+	+	−	−	−	−	−	−	4
3.2	+	+	+	−	−	−	−	−	−	2
3.3	+	+	+	+	−	−	−	−	−	2
3.5	+	+	+	+	−	−	−	−	−	2
3.6	+	+	+	+	−	−	−	−	−	3
3.7	+	+	+	+	−	−	−	−	−	1
3.8	+	+	+	+	−	−	−	−	−	2
3.9	+	+	+	+	+	−	−	−	−	4
4.0	+	+	+	+	+	+	−	−	−	3
4.1	+	+	+	+	+	−	−	−	−	1
4.3	+	+	+	+	+	+	−	−	−	3
4.5	+	+	+	+	+	+	+	−	−	2
4.7	+	+	+	+	+	+	+	+	−	2
4.9	+	+	+	+	+	+	+	+	−	2
5.2	+	+	+	+	+	+	+	+	−	2
6.0	+	+	+	+	+	+	+	+	+	2
6.3	+	+	+	+	+	+	+	+	+	4
7.1	+	+	+	+	+	+	+	+	+	4

*+=Ery. rhusiopathiae isolated; −=Ery. rhusiopathiae not isolated.

RESULTS

Samples of stomach contents from 164 different swine were examined in order to establish the range of pH values in apparently healthy animals. The pH value for stomach contents collected at the abattoir were similar to those collected by aspiration of stomach contents from normal live swine. The pH values ranged from pH 1.5 to pH 6.5; the mean value, pH 4.0; the median value, pH 3.9; and the mode, pH 4.5.

The results of culture following the addition of *Ery. rhusiopathiae* to the stomach contents are recorded (table 1). The length of time the organisms remained viable increased as the acidity of the stomach contents decreased toward neutrality (pH 7.0). The organism was recovered in three tests at pH 4.0, but not after a two-hour incubation, in the only test at pH 4.1. The organisms recovered had remained in the smooth phase.

The initial pH values of the soil ranged between pH 7.2 to pH 7.6. The organism recovered from the soil after inoculation was in the smooth phase. The result of the inoculation of soil in test tubes is recorded (table 2). The viability of the organism did not depend on whether the inoculum was 1 or 3 ml. The sterilizing of the soil from the farm resulted in maintenance of the organism for a longer period of time. The organism could at no time be recognized in soil by making stained smears.

The results of the isolation of the organism from the soil in the large clay pots are recorded (table 3). The pH values for the soil in the clay pots during the winter-spring test are given (table 4).

The pH of the soil was similar in the other tests.

The organism recovered from the soil was always in the smooth phase. The average temperature during the summer experiment was 70 F., the autumn temperature 55 F., and the winter and spring temperature 42 F. It was not unusual during the winter to find the soil frozen but, as spring thaws and rains developed, the soil was frequently soft and muddy. The dilution of the soil sample in saline solution did assist in the isolation of *Ery. rhusiopathiae*.

The drenching of the swine with the soil following winter and spring tests did not produce any infection. There was no rise in temperature for 14 days and blood cultures were negative. Total leukocyte and differential counts were within normal limits. The 2 animals drenched with soil from pot 1 and pot 2 were negative for gross evidence of *Ery. rhusiopathiae* infection on necropsy as well as on culture. These animals did not harbor the organism in their tonsils.

The 3 swine that were scratched and inoculated with strains of *Ery. rhusiopathiae* isolated from the soil all reacted with a rise in temperature, as in previous

TABLE 2—Viability of Erysipelothrix Rhusiopathiae in Soil in Test Tubes

Origin of soil sample*	No. of samples	Amt. of inoc.	Sterile soil		Nonsterile soil	
			Final pH	Days Ery. rhusio. isolated	Final pH	Days Ery. rhusio. isolated
Farm (1)	1	1 ml.	7.4	8	7.5	6
Farm (2)	1	3 ml.	7.4	8	7.5	6
Farm (3)	2	1 ml.	7.2	9	7.0	8
Farm (4)	2	3 ml.	7.2	9	7.2	8
Farm (5)	3	1 ml.	7.2	11	6.9	6
Farm (6)	3	3 ml.	7.2	15	6.9	6
Farm (7)	4	1 ml.	7.4	8	7.2	8
Farm (8)	4	3 ml.	7.2	8	7.2	8
Main sow pen (1)	5	1 ml.	7.2	4	6.8	8
Main sow pen (2)	5	3 ml.	7.2	4	6.9	8
Main sow pen (3)	6	1 ml.	7.2	7	7.3	10
Main sow pen (4)	6	3 ml.	7.2	7	7.3	10
Main sow pen (5)	7	1 ml.	7.5	4	7.5	9
Main sow pen (6)	7	3 ml.	7.5	5	7.5	9
Outer sow pen (1)	8	1 ml.	7.4	4	7.2	8
Outer sow pen (2)	8	3 ml.	7.4	6	7.2	8
Outer sow pen (3)	9	1 ml.	7.2	4	6.4	7
Outer sow pen (4)	9	3 ml.	7.2	5	6.8	9
Boar pen (1)	10	1 ml.	7.2	1	7.2	9
Boar pen (2)	10	3 ml.	7.0	4	7.3	10
Boar pen (3)	11	1 ml.	7.4	2	7.7	7
Boar pen (4)	11	3 ml.	7.4	5	7.7	10

*Each sample from different location in area.

TABLE 3—The Isolation of Erysipelothrix Rhusiopathiae from Pots of Soil

Postinoculation days	Summer test*						Autumn test*						Winter-spring test*		
	Pot 1		2		3		Pot 1		2		3				
	S	D	S	D	S	D	S	D	S	D	S	D	Pot 1	2	3
1	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
2	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
3	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
4	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
5	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
6	−	−	+	+	+	+	+	+	−	−	−	−	+	+	+
7	−	−	+	+	+	+	+	+	−	−	−	−	+	+	+
8	−	−	+	+	+	+	+	+	−	−	−	−	+	+	+
9	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+
10	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+
11	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+
12	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+
13	−	−	−	−	−	−	−	−	−	−	−	−	+	−	+
14	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+
15	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+
16	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+
17	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+
18	−	−	−	−	−	−	−	−	−	−	−	−	+	+	+
19	−	−	−	−	−	−	−	−	−	−	−	−	−	+	−
20	−	−	−	−	−	−	−	−	−	−	−	−	−	+	−
21	−	−	−	−	−	−	−	−	−	−	−	−	−	+	−
22	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
23	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−

*S-surface; D-deep (4 in. below surface); pot 1-sterile; pots 2 and 3-not sterile; summer test-1 inoculation; autumn test-2 inoculations; winter-spring test-6 inoculations.

experiments. The disease was not serious, but generalized skin lesions developed. The blood cultures were positive for two to three days. The animal that had been drenched with soil previously reacted to this inoculation, a further indication of the absence of the organism in the soil. The scratched, but uninoculated swine, did not show any reaction indicative of infection. It was refractory to a second inoculation with strains of *Ery. rhusiopathiae* obtained from the U.S.D.A.

Two of the 3 swine which were scratched and inoculated with soil organisms showed a synovitis in the carpal and tarsal joints and *Ery. rhusiopathiae* was recovered from these lesions. These 2 animals also had an agglutination titer of 1:400 and mouse protecting antibodies in the final serum.

Erysipelothrix rhusiopathiae was recovered from the tonsils of all 4 swine which had been exposed after scarification. However, bacteriological examinations of lymph nodes and organs were negative.

DISCUSSION

Digestion in the stomach of the hog was reported[11] to continue from one meal to the next although, in a moderate-sized meal, part of the gastric contents passed into the intestine three or four hours after eating.

TABLE 4—The pH Values for Soil in the Clay Pots During the Winter-Spring Test

	Before inoculation	Final pH
Pot 1	pH 6.9	pH 7.4
Pot 2	pH 7.4	pH 7.5
Pot 3	pH 7.3	pH 7.2

Since the mean pH value was pH 4.0, viable organisms may pass through the stomach into the intestine and as the pH increases the chance of survival increases. The results of this experiment indicate that the organism may be shed by the tonsil, remain viable in the stomach, and after intestinal passage may infect the soil.

The organism did not become established permanently in the soil used in this experiment, even when added in high concentration. In general, the organisms maintained their viability better in the nonsterile soil, which may indicate the possibility of some symbiotic relationship with other microorganisms. Certain soils in Nebraska were reported to be more suitable than others for the retention of *Ery. rhusiopathiae*.[14] In South Dakota, the organism showed no marked preference for certain soils although the more severe acute erysipelas occurred within areas of the heavier gumbo clay soils.[8]

No attempt was made in the present study to determine what other organisms were present in the soil, and it is possible that the soil used in our tests contained antagonists against *Ery. rhusiopathiae*. The soil was from areas in which swine erysipelas had occurred, and samples were

taken during the summer and fall when the majority of cases of acute swine erysipelas appeared. Therefore, it is thought that if the organism persisted in the soil for any length of time the necessary conditions should have been present.

The organism persisted in the soil longer during the winter and spring season, probably because of our inoculation technique and the preservation of the organism by low temperatures.

The results from drenching the swine with the soil indicated that either the organism was not present or it was not present in sufficient concentration to produce either carriers or immunity.

The strains of *Ery. rhusiopathiae* isolated from the soil at varying periods did not produce any appreciable difference in the zone of erythema in dermal scratches. The uninoculated animal probably contacted the organism at the time of scarification of the other 3, but not in sufficient numbers to produce clinical evidence of infection. The exposure may have been sufficient, however, to produce immunity, and to create a carrier.

The results of this experiment do not substantiate the general belief that the soil is an active vehicle for viable *Ery. rhusiopathiae* for a long period of time. It was demonstrated that when the organism was inoculated into the soil in large quantities it persisted for a relatively short period of time. Thus, it is possible that swine with acute erysipelas could infect the soil with a virulent strain for a sufficient period for it to be a source of infection.

It is believed, that to assure a constant source of infection from the soil, inoculation with large quantities of organisms must be carried out regularly. The suggestion that the soil is a frequent source of *Ery. rhusiopathiae* infection would appear not to be universally true.

SUMMARY

The stomach contents of normal swine at pH 4.3 and above did not destroy viable *Erysipelothrix rhusiopathiae* for at least three hours, and for longer periods as neutrality was approached. *Erysipelothrix rhusiopathiae* could be recovered from sterile and nonsterile soil for short periods of time when seeded on one or two occasions. Six inoculations of soil extended the period of recovery to a maximum of 21 days, but did not produce a permanent infection. Soil from areas where swine erysipelas has occurred may not become permanently infected with *Ery. rhusiopathiae* unless reinfected at regular intervals.

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Lime and the Soil

Although the need for limestone for soil improvement is as urgent as ever, much less is being used; thus profits are reduced. In Wisconsin, 6 million acres of crop land (60%) is acid and needs 3.5 tons of ground limestone per acre (as in eastern Iowa).

In Ohio, comparisons showed lowest yields of all crops where neither fertilizer nor limestone was added; next lowest, with fertilizer but no limestone; next highest, with limestone but no fertilizer; and best crops with both fertilizer and limestone.

Use of limestone must be correlated with other practices such as use of manure and fertilizer, growth of legumes, and proper rotation.

In Illinois, use of ground limestone has decreased from 5.5 million tons in 1946 to 2.5 million tons in 1955. Tests show that adding limestone alone returns $7.46 for each dollar invested in the limestone; $11.21 when used with fertilizer.—*Hoard's Dairyman (Feb. 10, 1958): 132.*

Defining the Hog Belt.—The Midwest hog belt includes the states of Iowa, Illinois, and Indiana, plus the adjoining parts of neighboring states; this includes five southeastern counties of Wisconsin.—*Wisconsin Agric. (Feb., 1958): 38.*

Passive Immunity to Erysipelas, in Pigs, Obtained from Colostrum

In Europe, it has been believed that pigs up to 10 to 12 weeks of age have a high natural resistance against erysipelas, and that vaccination before then will be ineffective due to the pigs' inability to produce antibodies. However, in the United States, vaccination of young pigs is often considered necessary.

Resistance in young pigs must come either from active immunity as a result of subclinical infection or passive immunity from colostral milk. In Germany, immunity was demonstrated in pigs from one experimentally infected sow, which developed arthritis, when the pigs were over 10 weeks old. In other litters, from a sow with arthritis, immunity of some pigs lasted into the fifth month. It was also demonstrated that pigs from 1 day to 6 weeks old, from nonimmune sows, could be infected percutaneously, intravenously, and orally. Thus, the frequent failures in the past to experimentally infect pigs with erysipelas may be explained by passive immunity in them.

It is generally understood that, in cattle, horses, and swine, maternal antibodies are transferred postnatally in colostrum, but not through the placenta, and that the intestinal wall is permeable, during the first few days of life, for antibody-carrying *gamma* globulin. Only *gamma* globulins from the protein group contain antibodies. Newborn pigs do not contain *gamma* globulin and their ability to produce antibodies starts slowly, later.

Newborn pigs, whether from immune or nonimmune sows, were found serologically to be without erysipelas antibodies. Also, 5 pigs from an immune sow, which were artificially fed without colostrum, were still serologically negative at 18 days, while pigs from negative sows which were fed antibody-containing colostrum showed immunity. One newborn pig, artificially fed 170 cc. of colostrum in several portions, had a lower antibody titer than did its litter mate nursing the same sow, probably because it ingested less milk during the first day or two. Young pigs fed blood serum from immune sows had lower antibody titers and immunity than did litter mates artificially fed colostrum from the same sow.

The duration of this passive immunity in young pigs is dependent on the level of the immunity of the sow, unless there is a deficiency of the colostral milk or poor absorption by the pigs' intestinal wall. When the immunity of pigs in a litter of an immune sow was challenged, those tested at 4½ weeks were immune; those at 8 weeks showed local skin reactions; and those challenged at 5 months developed generalized erysipelas septicemia. Results were similar in many litters.

With regard to young pigs developing long-lasting active immunity from ingesting the organism from excreta of the sow, along with colostrum, pigs left with the sows had no better immunity than did litter mates which were removed and artificially fed in sterile quarters after 4 days of age.

One susceptible sow which developed acute erysipelas, ten days after farrowing, had a high antibody titer a few weeks later. Her pigs were not separated from her, yet they remained serologically negative and developed generalized erysipelas when experimentally infected at 8 weeks of age.*

The explanation for the supposedly natural resistance of young pigs, in Europe, must be that most of the sows have developed immunity from natural exposure. The protein and *gamma* globulin contained in colostral milk passes from the body after

*A similar instance occurred in June, 1948, in northwest Iowa. Two sows in a herd died suddenly while nursing pigs about 2 weeks old. Necropsy and laboratory tests were positive for erysipelas for the second sow. None of the pigs sickened at the time, yet some of them developed acute swine erysipelas about six weeks later.—W.A.A.

three to six weeks, yet the immunity has been shown to last for several months.—*G. Wellman and F. Heuner in Zentralbl. f. Vet.-med., 4, (1957): 557 (trans. by Dr. R. D. Shuman, Beltsville, Md.).*

Colostrum and Antibodies in Pigs

Pigs are born devoid of antibodies against infectious diseases. They have been found to acquire the specific antibody titer in a few hours after ingesting colostrum from sows which were immune to influenza or to brucellosis. Pigs deprived of colostrum had no *gamma* globulin before 6 weeks of age, while those on colostrum had it a few hours after nursing. Likewise, antibody response to antigen was not detected until these pigs were 6 to 8 weeks old, while pigs which had ingested colostrum did respond to antigens.

Since young animals usually do not respond significantly to active immunization, the influence of age and colostrum on development of antibodies in baby pigs was investigated.

The 16 colostrum-deprived pigs did not respond with antibodies following the first injection of *Brucella abortus* bacterin, when 3 to 8 weeks old, but most of them did respond to a second inoculation three weeks later.

The 11 pigs nursing a sow from a brucellosis-free herd responded to the first inoculation, with antibodies, in four days (1 pig excepted). The second injection, three weeks later, produced severe anaphylactic shock in most of these pigs.

The 5 pigs which were allowed to nurse a sow given *Br. abortus* bacterin 14 days before farrowing (her colostrum titer was 1:400 for *Br. abortus*) had an appreciable *Br. abortus* titer (1:200) the first day, but the titer had decreased to 1:25 when they were 3 weeks old. These 5 pigs did not respond to the first inoculation of *Br. abortus* bacterin when 3 to 5 weeks old, but 4 of the 5 did respond with antibodies to the second inoculation three weeks later. All 5 pigs showed severe anaphylactic shock immediately after both inoculations. The 4 litter mates which were deprived of colostrum had no *Br. abortus* titer at birth or when 3 weeks old and did not respond to the first inoculation with bacterin but did respond to the second inoculation three weeks later.

These same pigs were similarly inoculated but at different times with ovine red blood cells, bovine serum, and egg white solution, with relatively similar results. While pigs deprived of colostrum were unable to produce antibodies until 8 weeks of age, those on colostrum showed responses to all of the antigens. While the response was coincident with the presence of serum *gamma* globulin, the antibody response was also affected by age; e.g., unlike the capacity for antibody formation, which increased markedly, the *gamma* globulin was highest during the first few days, then gradually decreased. Possibly some other factors in colostrum also affect antibody formation. Antibody formation is depressed in pigs on immune colostrum, thus it interferes with the immunizing of young pigs.

The anaphylactic shock was successfully treated with subcutaneous epinephrin and artificial respiration.—*A. B. Hoerlein in J. Immunol. (Feb., 1957): 112.*

Controlling Erysipelas in Europe

For ten years, in Germany, about 70 per cent of the swine herds have been vaccinated against erysipelas, but this has not affected its prevalence. Erysipelas is believed to be a noneradicable disease. Of the two methods used, simultaneous vaccination with a virulent culture produces a stronger and more prolonged immunity, but it has largely been supplanted by avirulent vaccines.

To test its ability to spread the disease, 5 ml. of a virulent erysipelas culture was mixed in the litter (bedding) in five pens containing 8 to 12 pigs each, on a large federal farm. Of the 46 pigs involved, 23 had been vaccinated and 23 were unvaccinated controls. The temperature of the litter was 27 C. (81.6 F.) and of the stable, 23 C. (73.4 F.). After three to five days, 3 vaccinated and 8 control pigs developed a septicemic form of erysipelas. When repeated on another farm with 29 vaccinated and 10 control pigs, using only 0.5 ml. of virulent culture, 1 vaccinated and 5 control pigs developed erysipelas, 4 of them as a chronic arthritis. None of the pigs in noninfected pens on either farm developed erysipelas.

In Poland, the avirulent (powdered) vaccine has been used for eight years. During 1949 and 1950, using a dose of 2 ml. per pig, erysipelas developed, respectively, in 0.2 and 0.3 per cent of 420,000 immunized

pigs and in 1.8 and 2.9 per cent of the 234,000 nonvaccinated control pigs. In 1951, when the dose was increased to 3.0 ml., only 0.1 per cent of the vaccinated pigs developed the disease. The immunity lasted at least three months.

On federal farms, where stable disinfection several times a year is compulsory, erysipelas is rare. Sanitation may be more important than vaccination.

It has been proved that biting insects (flies, lice) can spread erysipelas. Temperature may also be a factor; at room temperature, erysipelas is easily transmitted from sick to healthy mice, yet at less than 12 C. (53.6 F.), contact infection did not occur. Strongyloides larvae passing through the skin of pigs can carry the disease. The skin, as a "gate for the infection," seems to play an important role.

In Bulgaria, erysipelas is especially prevalent in May and June and again in October and November. In the past three years, an avirulent vaccine made of a Bucharest strain, injected once or twice, has resulted in a strong, long-lasting immunity.—*A. Stryszak in Monatsh. f. Vet.-med. (Aug. 15, 1957): 443.*

Production of Erysipelas Serum

The following items are from the "Yearbook of the Pasteur Serum and Vaccine Institute," vol. 2, Bucharest, 1957.

From horses.—Hyperimmunization of horses against swine erysipelas infection results in many deaths, especially in older horses. There seems to be no correlation between the serum titer and the development of arthritis and endocarditis. Titrating the serum by inoculating animals is inaccurate, more so in pigeons, in which resistance varies with the season, than with mice.—*V. Tomescu et al., page 29.*

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From swine.—Production of a satisfactory anti-swine erysipelas serum from hyperimmunized swine is not difficult and losses are less than with horses. However, the titer is less constant than in serum from horses, but it is more slowly eliminated from the inoculated animal, making it relatively more effective.—*V. Tomescu et al., page 40.*

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From cattle.—When 10 cattle hyperimmunized for the production of anti-swine erysipelas serum were slaughtered, 25 days after the last inoculation, *Erysipelothrix rhusiopathiae* was isolated in 9 of the 10 from the spleen, bone marrow, kidneys, or endocardial vegetations. The pathogenicity of these organisms was always reduced from that of the inoculum.—*D. Tacu, page 386.*

Acute Erysipelas and Overheating

On a hot day in August, 1956, an Illinois farmer lost 18 of his 150 hogs, averaging about 220 lb., and many more were sick. Believing that the trouble was from overheating, he sent a truckload of the healthy animals to market. The remainder, including the sick ones, were treated with anti-erysipelas serum. Nearly all of the latter recovered, whereas those sent to market were sick or dying on arrival.—*L. T. Boley in Auburn Vet. (Fall, 1956): 21.*

[Similar heavy losses were observed in extremely hot weather in several herds in Plymouth County, Iowa. On two occasions, in herds of several hundred healthy shoats which were on pasture with inadequate shelter from the hot sun, the owners frantically attempted to provide better shelters for the animals, only to find that losses continued. In both cases, the laboratory-confirmed diagnosis was acute, septicemic erysipelas, which was brought under control by the liberal use of penicillin and anti-swine erysipelas serum for the sick shoats and serum with culture for the well animals.—W.A.A.]

A Concentrated Anti-Hog Cholera Serum.—A new anti-hog cholera product, a concentrated blood fraction with twice the antibody content of the usual commercial serum, has been placed on the market by Armour Veterinary Laboratories under the name of Antrate H.C. It can be used with virulent or modified virus or alone for temporary immunity against cholera.

It is derived from hogs hyperimmunized against cholera but may contain antibodies for other diseases as well.—*Wallace's Farmer (March 1, 1958): 86.*

In an experiment, at the Florida Agricultural Experiment Station, rations containing chlortetracycline were preferred by swine to those containing oxytetracycline or penicillin V, whereas the animals avoided feed containing erythromycin when other feed was available.—*J. Anim. Sci. (Feb., 1958): 42.*

Present Status of Prophylactic Vaccination in Swine Erysipelas

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MY FIRST EXPERIENCE with swine erysipelas was about 1918, with a specimen sent from Illinois for diagnosis.* It was the spleen from a pig in a herd where the animals were dying after being noticed sick for only a few hours, or not at all. The organism isolated from the spleen was new to us. It fit the description of the swine erysipelas bacillus but, since this disease was not being recognized in this country at the time, we were reluctant to report it.

In 1922, after publication of an article on arthritis in swine,[1] we sent this culture to the authors and it was identified as a typical swine erysipelas culture. At that time, we knew little about how to control the disease. We had no specific biological agents and much reliance was placed on anti-hog cholera serum. Actually, many cases were diagnosed as hog cholera; however, recoveries were frequent in such herds, which is good evidence that it was not all cholera.

We now know that calcium deficiency predisposes pigs to erysipelas infection. Erysipelas organisms thrive in certain types of alkaline soils and plague each pig crop year after year. However, certain types of clay soil and acid soil, in general, are less favorable to the organism, and recurrence of disease year after year is not so common on this type of farm.

CONTROL OF SWINE ERYSIPELAS

The first approach to the control of swine erysipelas was through the use of autogenous bacterins made from cultures isolated from early acute cases. These bacterins seemed of value in some sections, but not in the badly infected areas of Nebraska. However, it soon became apparent that we were getting false impressions from the use of bacterins, as the disease in some herds cleared up without any treatment. Also, we know now that the low concentration of organisms used at that time would be of little value.

When anti-swine erysipelas serum became available, in 1931, it was used to stop the progress of the disease in a herd. However, the passive immunity was so short that acute erysipelas might recur in the same herd several times before the hogs reached market age, and the repeated medication of the entire herd was expensive and difficult. Therefore, it became general practice to treat only the visibly affected animals with serum. Not until about ten years later did the simultaneous method of vaccination with virulent culture and serum come into vogue.

Before virulent culture was available, we tried avirulent culture in the badly infected territory in Nebraska. The results were more favorable than with the bacterin but were still far from satisfactory; therefore, these methods were abandoned in favor of the officially released virulent culture vaccine used with serum.** By that time, many farmers in the eastern portions of Nebraska and South Dakota were literally being forced out of the hog business.

At first, no standard test for potency of anti-swine erysipelas serum existed, and each producer had his own interpretation of what constituted a potent serum. When these serums were used with the virulent *Erysipelothrix rhusiopathiae* vaccine, it was at once apparent that some were much more potent than others and that serial lots varied in potency. This meant variable results, frequently terminating in chronic cases from vaccine infection. A standard test was, therefore, prescribed, and a standard serum was supplied producers for comparative testing of all anti-swine erysipelas serum—a method still in vogue. The commercial product must protect as well as, or better than, the government standard when vaccinated mice or pigeons are exposed to a culture of sufficient virulence to kill all controls within the required time.

The potency of this standard serum has been increased as methods and techniques were developed to increase the efficiency of hyperimmunization of animals.

As far as I am aware, all anti-swine

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*Sent to the Kinsley Laboratory, Kansas City, Mo.

**The late Dr. L. Van Es, University of Nebraska, was the instigator of this project,[2] based on his experience in Europe before coming to this country.

erysipelas serum on the market today is prepared from the blood of horses hyperimmunized with cultures of *Ery. rhusiopathiae suis*. Other animals may be used for serum production, but horses are the most desirable as far as economics and ease of handling are concerned.

Originally, the use of virulent culture erysipelas vaccine was controlled through a "Memorandum of Understanding" between the various states and the U.S. Department of Agriculture. The project was set up as a controlled experiment and, to begin with, the vaccine was produced and distributed by the State of Nebraska and the U.S. Department of Agriculture laboratory at Lincoln, Neb.†

After the value of the project became apparent and the demand for vaccine increased materially, the production of the vaccine was turned over to commercial laboratories, in 1942, under a limited license. This limited license arrangement continued until July 1, 1957, at which time the experiment was terminated and the license was changed to a "special" basis.

A "special" license requires that a special permit must be obtained from the proper livestock sanitary official of each state into which the product is shipped or in which it is sold.

Many states (20) did not participate in the Memorandum of Understanding, usually because the officials objected to the use of virulent organisms in their state, or they considered the disease of insufficient importance in the state to justify the use of the vaccine, with its possible danger of contaminating farms where used.

In recent years, bacterins and avirulent vaccine have been made available for use by veterinarians. These have been a great advantage in states where the virulent culture was banned. Also, these products have made much headway in replacing virulent culture in some areas.

AN ERYSIPELAS CONTROL SURVEY

Recently, to determine the methods used by veterinarians in the control of swine erysipelas today, a questionnaire was sent to a group of 308 general practitioners, selected at random from the "AVMA Directory," in four states. From the 220 questionnaires returned (Illinois, 60; Iowa, 71; Missouri, 46; Nebraska, 43), the following information has been tabulated and assimilated.

Information requested on the questionnaire was directed to find out:

1) Which control product had been used by the veterinarian (some had used all of them at one time or another).

2) The one giving best results (left blank by many).

3) Whether "breaks" occurred following its use (this varied considerably).

4) What product he is using now (often more than one).

5) Which product he prefers (27 did not indicate a preference, 57 indicated more than one).

Erysipelas bacterin polled the highest preference rating (85). Also, it is being used by more veterinarians (100) than any of the other products. Breaks were reported by 106 of the 175 users (60.6%).

The simultaneous use of virulent *Ery. rhusiopathiae* vaccine and anti-swine erysipelas serum ranked second in preference (62) and is being used by 95 veterinarians. This method has been used by the most veterinarians (191), and they reported the highest percentage of breaks (141 or 73%). However, this procedure has been in use much longer than any, except serum alone, and it has had more time to show its shortcomings. Also, a common comment was that breaks following this procedure were not as severe as after some other methods.

Avirulent erysipelas vaccine alone ranked third in preference (49) and was being used by 66 veterinarians. Breaks were reported by 89 of the 147 who have used it (60.5%), almost the same percentage of breaks as with the bacterin.

Anti-swine erysipelas serum alone ranked fourth in preference (19) and had been used by 189 of the 220 veterinarians reporting. A few veterinarians use no other biological product for erysipelas control.

The simultaneous use of avirulent erysipelas vaccine and the serum was next (5th), with 17 preferring it. This combination had been used by 107 veterinarians, with 41 breaks (38%), and 28 are using the method now.

Of the 87 who had used anti-hog cholera serum to combat swine erysipelas, breaks were reported by 28 (32%).

Answers to the question "What age do you prefer to vaccinate pigs against erysipelas?" showed considerable variation.

†The virulent culture was used in Nebraska in 1940 and was released for use, under strict federal and state supervision, to Iowa and South Dakota in 1941.

Some might vaccinate at an early age on some farms, but preferred to let pigs get older for routine vaccination.

While the age preferred for vaccination was difficult to tabulate, the following indicates the preference trends of the 220 veterinarians: vaccinate at 1 to 3 weeks (36 veterinarians); at 4 to 5 (38); at 5 to 6 (32); at 6 to 8 (79); at 8 to 10 (33); at 12 weeks (1); varies (12). Some overlapped, but approximately half of the veterinarians preferred the range of 5 to 8 weeks of age in which to vaccinate pigs against swine erysipelas.

To the question "Do you favor vaccination of sows prior to farrowing to furnish a colostrum immunity to pigs?" 184 (84%) answered "yes." A number said "no"; others reported that it had failed to protect pigs.

The "remarks" were enlightening. An attempt has been made to summarize them according to their reference.

To begin with, 35 (16%) indicated that we still do not have a satisfactory immunizing agent or procedure with which to combat swine erysipelas.

Quite a few (20) used one product on young pigs, another on older ones. A like number indicated they varied the product according to the history of the disease on a particular farm.

A few (13) discriminated against products because they were available to the farmer. Similarly, many preferred products because they could not be purchased by the farmer.

More favorable comments were directed at erysipelas bacterin than any other product. Typical comments were "less trouble with bacterin," "more uniform results," "easy to give" (57). Others (28) liked the bacterin because it could be used at the time of vaccination for hog cholera.

Avirulent vaccines, likewise, drew a considerable number of similar comments (48), with a few (8) stressing the longer immunity resulting from this type of product. Avirulent vaccines are also used at the time of vaccination for hog cholera (by 9).

There were many favorable comments regarding the virulent vaccine, such as "less trouble with virulent culture and serum," "longer immunity," and "preferred in baby pigs" (56). A few (9) preferred this method in herds affected with acute erysipelas.

Many favored the vaccination of sows prior to farrowing. This procedure, which has resulted in a marked reduction in baby pig vaccination, is based on the finding[2] that pigs nursing erysipelas-immune sows carried immunity derived from the sow's milk until they were about 6 weeks old. If these immune pigs were vaccinated with erysipelas culture during this early age, they derived little or no benefit from it. The culture was literally blanked out, the same as would happen when too much serum is used with culture.

Many veterinarians have modified the original procedure, of vaccinating sows at or prior to breeding time, by vaccinating them four to six weeks prior to farrowing. This allows use of the products which do not produce as long-lasting immunity as the virulent vaccine and serum used by the government workers in the original experiment.

Experience has shown that an increased dose of vaccine in relation to serum was better than the original dose of 0.25 cc. of culture and 5.0 cc. of serum. Also, shock is common in some herds when the 5.0 cc. of serum is used on baby pigs. This has prompted many veterinarians to reduce the serum dose to 3.0 or 4.0 cc. with 0.25 cc. or more of culture.

We can now understand why the use of virulent culture with serum in baby pigs was followed by breaks before market age. This problem increased with the increased percentage of sows which carried some immunity from natural exposure or from vaccination. In the early days of vaccination, the sow was commonly ignored, but we are now utilizing the principles of immunity in a more practical way and on a mass basis.

Some veterinarians, who believe the immunity which follows the use of virulent culture and serum is better and longer lasting, use it only on certain farms because the bacterin or avirulent vaccine can be handled with less danger to the operator; also, they can be used simultaneously with hog cholera vaccination, whether it be with virulent virus or modified live virus vaccines.

The paper work incident to use of the virulent vaccine was a frequently mentioned reason for using other products. The fact that bacterins and avirulent vaccines do not spread infection was a prominent recommendation for them. However, a few (4) users stated that bacterins had been a complete failure in their hands, as had all avirulent vaccines for one or more users.

A few comments have not been quoted,

but the preceding cover the highlights of the information furnished by the practicing veterinarians.

The question of using a dose of vaccine with serum, followed one or two weeks later with a dose of vaccine alone, was raised by a few veterinarians (5). This was the original recommendation for use of virulent culture with serum.[3] However, it was demonstrated in the field that the second dose of vaccine did not produce any better results than the one dose with serum. Using control herds, as many breaks occurred in those given two doses of culture as in herds given one dose.

In an animal with some immunity, it is apparent that if the first dose of live culture had stimulated the pig to produce immunity at all, the second dose given a week or two later would be blanked out.

Conclusion and Summary

Anti-swine erysipelas serum alone is a potent product that will produce a passive immunity; however, protection can not be depended upon for more than two or three weeks. This may vary with the dose and with the individual animal. Also, this serum is derived from horses and, if pigs are given repeated doses at intervals of a few weeks or months, they may become sensitized and fatal shock may occur after the third or fourth dose.

The virulent vaccine and serum will probably confer the most solid and long-lasting immunity of any of the products available, provided that the pigs are susceptible and that the dose of serum is small or marginal. Of course, a healthy pig is a prerequisite with all of the immunizing agents.

The avirulent vaccines can not be used in animals showing immunity, for the same reasons given for the virulent products.

Avirulent erysipelas vaccines have been used in Europe for some time and are still used in some countries. They are considered effective in producing an immunity of about three months' duration, but not much more.

Erysipelas bacterin will produce a favorable response under more variable circumstances and is effective in stimulating an immunity which usually lasts for about three months. A second dose of bacterin, two to four weeks after the first, materially increases the titer of the animal and markedly prolongs the immunity.

Remember this—certain individuals will not react to the usual vaccination procedure, and will remain susceptible to disease regardless of the product used. Fortunately, these are rare.

The principal drawback to using bacterin is its irritating effect on local tissue. However, this is a part of the process which is needed to produce the desired results. The point of injection is important, to avoid involvement of nerves or swelling where it will interfere with locomotion.

The use of antibiotics is an established procedure in treating animals infected with erysipelas. A combination of antibiotic and anti-swine erysipelas serum is better as a curative procedure than either of these agents alone.

The question has been raised about the effect of antibiotic-supplemented feeds on reactions of vaccines in pigs eating such feeds. We have no information on this that I know of.

This discourse should not be concluded without reference to the human infections that may develop either from accidental vaccination or exposure to infection at autopsy of affected swine or turkeys. It behooves the veterinarian to keep advised on how to handle such problems where diseases of animals are transmitted to man. Many local physicians are not familiar with erysipeloid, which is the human infection caused by this organism.

The use of antiserum is not recommended in man, because of the possible bad side effects. However, antibiotics are effective. If you know you are exposed, it is advisable to counteract the infection before symptoms appear. Many times it is completely controlled, without any evidence of its presence, if handled in this manner. Penicillin is effective in this way.

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Erysipelothrix Rhusiopathiae Infection in Joints from Market Hogs in Canada

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THERE IS EVIDENCE that the majority of cases of arthritis in swine are caused by *Erysipelothrix rhusiopathiae*. In the United States, from 1945 to 1952, arthritis in swine ranked second or third as a cause of condemnation of swine carcasses at meat-packing plants under federal veterinary inspection.[1]

A review[6] of hog condemnations in Canadian meat-packing plants, from 1929 to 1950, for conditions that could be *Ery. rhusiopathiae* infection showed an increased incidence of arthritis—the rise being sharpest after World War II. However, the reports of the Veterinary Director General of Canada[2,3,4] indicate that chronic swine erysipelas had not increased from 1952 through 1954; the total condemnation due to arthritis remaining at 42 to 43 per cent.

LITERATURE CITED

In the United States, the isolation of *Ery. rhusiopathiae* from the joints of 1 pig was reported in 1919 after a disease, which was not recognized, had passed through a drove of 120 pigs, 30 of which had died.[14] The isolation of this organism from a pig with polyarthritis was reported in 1922.[21] In 1933, *Ery. rhusiopathiae* was first observed as the cause of acute swine erysipelas in Canada.[20]

Surveys have been conducted in various countries to determine the incidence of the isolation of *Ery. rhusiopathiae* from joints of pigs affected with arthritis.[8,9,11,19,22] In a report from the United States (1942), out of a total of 572 arthritic joints, this organism was recovered from 444 or 77.6 per cent.[26] The percentage might have been higher had it been practical to make extensive cultural tests of the affected joints.

In Canada, *Ery. rhusiopathiae* was reported (1952) to have been isolated from 36.5 per cent of the arthritic joints from 458 hogs.[6] In a report from Nebraska (1942) the hip, tarsal, carpal, and phalangeal joints were most frequently involved.[29] However, no joint appears to be exempt, and all joints, even those of the spinal column, have been found enlarged.[12]

The infection was said to disappear from affected joints of some swine while others remain infected.[8] In joints becoming sterile, remission, resolution, and complete recovery might occur and connective tissue increases might lead to adhesions and ankylosis. The synovitis might assume certain benign, neoplastic properties, and might persist and extend in the absence of the primary cause. It was emphasized that the arthritis in swine erysipelas was a polyarthritis—a chronic form of the infection.

In a more recent study of the experimental disease,[17,18] *Ery. rhusiopathiae* could not be isolated from swine with advanced chronic arthritis more than 226 days after exposure. The pathological changes in the joints of pigs with chronic arthritis, in a study of the experimental disease in Indiana in 1957,[19] were, in general, similar to those from England in 1940.[8]

An infectious agent which produced arthritic lesions indistinguishable from those of erysipelas was isolated in Iowa;[13] *Ery. rhusiopathiae* was recovered from only about a quarter of the cases of arthritis in young pigs. Evidence against *Ery. rhusiopathiae* being of etiological significance in swine erysipelas was also produced.[7]

MATERIALS AND METHODS

The joints examined in this survey were secured by veterinary meat inspectors employed by the Federal Department of Agriculture (Canada). Joints were removed, at the time of slaughter, from market hogs that were classified as cripples or considered to have arthritis. Only one joint was removed from each hog.

On arrival at the laboratory, the joints were skinned and wrapped individually in waxed paper. At the time of culture, synovial fluid was withdrawn aseptically with a Pasteur pipette, and 0.2 ml. was inoculated on the surface of trypticase soy agar* containing 5 per cent sheep blood. Incubation was aerobic at 37 C. for 24 to 36 hours. If sufficient synovial fluid was not obtained, a sterile glass rod was rubbed over the synovial membranes and transferred to the surface of the agar.

After the cultures were completed, the joints were returned to the refrigerator. If *Ery. rhusiopathiae* was obtained, the joint was opened and gross observations made; when it was not isolated, the affected joint was recultured. After three negative cultures in a ten-day period, the gross pathological changes were observed. This procedure, of three attempts at culture, was abandoned half way through the project when it became obvious that it did not increase the number of isolations. *Ery-*

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TABLE 2—Results and Percentages of Culture and Alterations Found in Joints of Crippled or Arthritic Market Hogs

Joint and culture results	Total		Gross alterations in joint and synovium												Growth of Ery. rhusiopathiae**					
			Active inflammation		Inactive inflammation		Calcified		Diffuse hyperplasia		Focal hyperplasia		Copious synovial fluid		Poor		Fair		Good	
	(No.)	(%)	(No.)	(%)	(No.)	(%)	(No.)	(%)	(No.)	(%)	(No.)	(%)	(No.)	(%)	(No.)	(%)	(No.)	(%)	(No.)	(%)
Elbow	34																			
pos.*	17	50.0	15	88.2	2	11.7	4	23.5	12	70.6	2	11.7	4	23.5	6	35.3	4	23.5	7	41.2
neg.*	17	50.0	10	58.8	7	41.2	1	5.9	9	52.9	2	11.7	6	35.3	..	....	..	....	..	....
Metacarpal	2																			
pos.	1	50.0	1	100	..	....	1	100	1	100	..	....	1	100	..	....	..	....	1	100
neg.	1	50.0	..	....	1	100	..	....	..	....	1	100	..	....	..	....	..	....	..	....
Metacarpo-phalangeal	205																			
pos.	142	69.2	126	88.8	16	11.2	88	61.9	130	91.5	5	3.5	3	2.1	54	35.2	19	13.3	69	48.6
neg.	63	30.7	19	30.2	44	69.8	46	73.0	41	65.1	..	....	..	....	..	....	..	....	..	....
Stifle	185																			
pos.	125	67.5	120	96.0	5	4.0	9	7.2	115	92.0	10	8.0	59	47.2	61	48.8	22	17.6	42	33.6
neg.	60	32.4	45	75.0	15	25.0	1	0.8	39	65.0	12	20.0	25	41.7	..	....	..	....	..	....
Tarsal	137																			
pos.	73	53.2	68	93.1	5	6.8	21	28.8	67	91.8	3	4.1	10	13.7	32	43.8	18	24.6	23	31.5
neg.	64	46.8	29	45.3	35	54.7	14	21.9	29	45.3	15	23.4	6	9.4	..	....	..	....	..	....
Positive	358	63.5	330	92.2	28	7.8	123	34.3	325	90.8	20	5.6	77	21.5	153	42.7	63	17.6	142	39.6
Negative	203	36.5	103	50.2	102	49.8	62	30.2	118	57.5	30	14.6	37	18.0	..	....	..	....	..	....
Total	563	.....	433	76.9	130	23.0	185	32.8	443	78.7	50	8.9	114	20.2	..	....	..	....	..	....

*Pos.—Erysipelothrix rhusiopathiae isolated; neg.—Ery. rhusiopathiae not isolated. **Poor—1 to 10 colonies; fair—10 to 20 colonies; good—more than 20 colonies.

TABLE 1—Erysipelothrix Rhusiopathiae Infection in Joints of Market Hogs at Time of Slaughter

	Crippled hogs slaughtered		Hogs with arthritis at slaughter		Ery. rhusiopathiae isolated			
					Joints		Hogs slaughtered	
Submission	(No.)	(%)	(No.)	(%)	(No.)	(%)	(%)	Total
1	13	0.5	42	1.8	29	69.0	1.2	2,363
2	19	0.4	55	1.3	31	56.3	0.7	4,275
3	12	0.4	53	1.6	38	71.7	1.1	3,310
4	16	0.7	55	2.3	38	69.1	1.6	2,363
5	21	0.4	60	1.2	34	56.7	0.7	4,655
6	54	1.3	63	1.5	37	58.7	0.8	4,292
7	39	1.0	57	1.4	33	57.9	0.8	4,062
8	16	0.4	96	2.5	63	65.6	1.6	3,882
9	10	0.2	64	1.6	45	70.3	1.1	3,882
Total	200	0.6	545	1.5	348	63.8	1.0	34,969

sipelothrix rhusiopathiae was identified on the basis of colony morphology, its reaction in a gelatin stab culture, and a smear stained by the Gram method.

A general examination was made for evidence of active inflammatory arthritis, based on the presence of an engorged, edematous, diffuse or focal, hyperplastic synovial membrane, with or without an increase in synovial fluid. If synovial fluid flowed freely on incision of the joint capsule, it was designated as copious. Vascularity was a significant criterion of an active process.

The arthritic process was considered inactive when there was no congestion or edema of the synovial membrane, although a diffuse or focal thickening could be present. This thickening was avascular, and alterations indicated that the pathological process was undergoing remission or recovery.

RESULTS

The incidence of *Ery. rhusiopathiae* infection in joints of the hogs in this study is recorded (table 1). This table contains information only on joints which were obtained from sources where data were available on the number of hogs slaughtered and on the number of those with arthritis that were cripples.

The term "submission" refers to the shipment of joints received in the laboratory. It was not known the days of slaughter this represented—presumably, each submission of joints varied as to the number of days. The greatest number of cripples slaughtered in one submission was 54; an additional nine arthritic joints were submitted at that time. Of the 63 joints, 37 (58%) yielded *Ery. rhusiopathiae*. The least number of cripples was 10, but at that time 54 other arthritic joints were received. *Erysipelothrix rhusiopathiae* was isolated from 45 (70%) of these joints.

The results (No. and %) of culturing and the gross pathological changes found in joints of crippled or arthritic market hogs are listed (table 2, opposite page). Of the 563 joints included in this table, 18 were not included in table 1 since they were not accompanied by information on cripples or number of swine in the slaughter.

The carpal and stifle joints were most frequently submitted and *Ery. rhusiopathiae* was isolated most often from these articulations. The two metacarpophalangeal joints submitted were enlarged, and one had an active process and contained a copious amount of synovial fluid.

Of the 137 tarsal joints, 73 (53.2%) yielded *Ery. rhusiopathiae* and growth was good in only 23 (31.5%) of the isolations. There was more calcification of joints in the front legs than in the hindlegs. This was especially true in the area of the carpal joints. Calcification as observed throughout this survey varied from osteophyte formation to complete ankylosis. In cases of complete ankylosis, which were not common, the isolation of *Ery. rhusiopathiae* became difficult and growth was poor.

The synovial membrane of 154 stifle joints showed extensive diffuse hyperplasia. It was common in the 39 synovial

TABLE 3—Bacteria Isolated in the Absence of Erysipelothrix Rhusiopathiae from Joints with Active Inflammation

Joint	No.	Species of bacteria
Elbow	1	Hemolytic streptococci
	2	Escherichia coli
	2	Corynebacterium pyogenes
Carpal	1	Pasteurella multocida
	1	Escherichia coli
Stifle	2	Pseudomonas aeruginosa
	2	Escherichia coli
Tarsal	2	Proteus vulgaris
	1	Pseudomonas aeruginosa
Total	14	

membranes with diffuse hypeplasia, from which *Ery. rhusiopathiae* was not isolated, to observe nodules that appeared neoplastic.

The bacterial species which were isolated from joints with active inflammatory changes in the synovial membrane are tabulated (table 3). The elbow contained multiple large abcesses when *Corynebacterium pyogenes* was isolated. The abscesses were joined by fistulous tracts which extended into the eroded and necrotic articular surfaces. A copious amount of synovial fluid was present when *Pseudomonas aeruginosa* and *Proteus vulgaris* were isolated.

Hemorrhage was found in the stifle on two occasions. In one case, there was no evidence of a synovitis whereas, in the other, an acute synovitis was present and *Ery. rhusiopathiae* was isolated. It is considered that the hemorrhage in the former case was due to trauma at the time of slaughter.

DISCUSSION

It is realized that the number of joints selected by veterinary inspectors did not represent the total number of hogs suffering from arthritis. In some cases, to diagnose arthritis, it was necessary for the veterinarian to incise the joint capsule, thus eliminating the possibility of culturing it. In addition, some hogs in the early stages of, or undergoing remission from, arthritis were probably overlooked.

A variation in the percentage of animals classified as cripples in the submissions is listed in table 1. This variation may be due to procedures followed by meat packers in accumulating a number of cripples, then slaughtering cripples at one period in the kill, or to the arrival at market of a large number of cripples.

It is of interest that 1.5 per cent of the hogs represented in part of this survey showed arthritis and 63.8 per cent of the 545 arthritic joints yielded *Ery. rhusiopathiae*. The 1 per cent infection of 34,969 hogs is a minimal percentage. Some arthritic cases were missed and *Ery. rhusiopathiae* may not have always been recovered when present. Inoculating mice with joint fluids might have increased the recoveries. The failure to isolate *Ery. rhusiopathiae* from cripples may indicate that cripples represented a prolonged chronic state and the organism had disappeared but the arthritis continued in the absence of the primary cause.

Not only were more carpal and stifle articulations submitted (table 2), but the percentage of infection in these joints was greater than in the others examined. The growth of *Ery. rhusiopathiae* isolated from the carpal joint was better than that from the stifle joint; 69 (48.6%) of the infected carpal joints, but only 42 (33.6%) of infected stifles, yielded the organism in good quantity.

It is believed that this is related, in part, to the amount of synovial fluid, as 59 (47.2%) of infected stifle joints contained a copious amount of fluid. It had been observed that, as the amount of the synovial fluid increased, the growth of *Ery. rhusiopathiae* decreased. This may be due to the presence of antibody in the synovial fluid, as serum antibody titers against *Ery. rhusiopathiae* increase in chronic arthritis.[15]

The increase in synovial fluid in the stifle joint may be to aid mobility of the joint. The carpal joint is more exposed to physical trauma. Such trauma in the presence of a synovitis could further damage the periosteal attachments and permit the development of an osteophyte, eventually extending to complete ankylosis.

This may be the reason for a higher incidence of calcification in the front legs, especially in the carpal joint. The lower number of isolations from the tarsal joint may be the result of an inability to open and expose the synovial membrane, as compared to the relative ease of exposure in other joints.

The isolation of *Ery. rhusiopathiae* from joints in which the inflammatory process did not appear active (thus designated inactive) should not imply that the organism was present as a saprophyte. The observation of inactivity of the inflammatory process in the synovial membrane may be because of a remission, an early infection, or a failure to observe focal areas of hyperplasia and vascularity.

The demonstrations of infection with *Ery. rhusiopathiae* in 63.5 per cent of 563 joints is greater than the 36.5 per cent previously reported from Canada,[6] but not as great as the 444 (77.6%) in 572 arthritic joints found in hogs from 16 states, the majority of which were from the corn belt, an area of high porcine population.[16]

The recording of pathological alterations

is considered a useful adjunct to bacteriological examination. It reveals possible inadequacies of the cultural techniques. The gross changes relate the stage of the disease process and may be of assistance in explaining the failure to isolate *Ery. rhusiopathiae.*

In this survey, a total of 433 joints showed signs of active inflammatory processes and, of this number, 330 (73.9%) yielded the organism. Fourteen of the joints with active inflammation yielded a variety of microorganisms in the absence of *Ery. rhusiopathiae* (table 3). These organisms may be secondary invaders, contaminants, opportunists, or may result from a septicemia or bacteremia.

SUMMARY

A survey of 563 joints from crippled or arthritic market hogs is reported. *Erysipelothrix rhusiopathiae* was isolated from 358 (63.5%) of these joints. This was a 1 per cent incidence of infection with *Ery. rhusiopathiae* in the 34,969 hogs slaughtered.

The carpal joint was examined most frequently; 142 (69.2%) of the 205 joints yielded *Ery. rhusiopathiae* and 69 (48.6%) gave good growth. Calcification occurred in 88 (61.9%) of infected carpal joints; more often than in other joints. Of the 185 stifle joints, 125 (67.5%) yielded cultures of *Ery. rhusiopathiae;* however, growth was poor in 61 (48.8%) of the cultures. A copious amount of synovial fluid was present in 84 stifle joints. The elbow, metacarpophalangeal, and tarsal joints were examined also; however, *Ery. rhusiopathiae* was not isolated as often as in the stifle and carpal joints.

The gross pathological changes are tabulated (table 2) on the basis of inflammatory changes observed in the joint and synovial membrane. Active inflammatory changes were based on degree of vascularity, edema, and hyperplasia of synovial membrane. Of the joints examined, 433 (76.9%) were classified as possessing active inflammatory changes.

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Radiation Biology and the Veterinarian

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VETERINARIANS are looking optimistically to the future as an era of achievement and of advancement in all phases of the profession. The purpose of this paper is to present some of the possibilities for veterinary medical research in the field of radiation biology.

The modern veterinarian must be prepared to assist civilian populations in the event of a nuclear disaster. He is important on the civil defense team because of his knowledge of medicine and surgery and his ability to assist physicians in the first-aid requirements following such a disaster. His contribution in determining the suitability of foods for human consumption may, however, be of greater importance to the over-all recovery program.

Radioactive fall-out can be more dangerous to livestock than to human beings. It is assumed that people routinely wash all fruits and vegetables before eating them and remove much of the surface fall-out contamination. Livestock, on the other hand, graze over large areas, consuming contaminated forage. Dairy cows subsequently secrete an appreciable amount of some of the fission products in the milk, and they eventually are found in other dairy products. A small amount of the fission product may be stored permanently in the bones of livestock. What proportion of ingested radioactive material is found in edible meat products is still being investigated. Studies of this sort, employing experimental animals, will provide a particularly promising field for the research veterinarian.

Studies in the field of radiation biology are complex, and a considerable knowledge of chemistry, physics, mathematics, and other basic sciences is needed to comprehend and to interpret some of the phenomena observed. Although few of these subjects are included in the regular veterinary and medical curriculums, training courses in radiobiological techniques are being offered in a number of laboratories. The addition of such training to the veterinarian's basic education in the normal physiology, anatomy, and diseases of farm and laboratory animals will make him a valuable member of a research team.

APPLICATION OF RADIATION BIOLOGY

Since Danlos and Block first used the ionizing radiation from radium for therapeutic purposes, medical science has sought other substances which would be more specific in their selectivity and uptake by body tissues. Such selectivity would decrease the over-all radiation burden borne by the body as a whole. Physiologically, many elements are known to concentrate in certain organs, and with the preparation of radioisotopes of these elements, a truly specific system of irradiation becomes available. From over 100 known elements, some 800 radioisotopes have been prepared, of which slightly over 100 are available from nuclear reactor preparations. Radioiodine is a prime example of an isotope which concentrates in a specific tissue—the thyroid gland. Because of the nature of its radiations, the greater part of an administered dose of I^{131} does not escape beyond a few millimeters from the surface of the gland. This concentrating ability has made it possible to use large doses of radioiodine in treating certain forms of thyroid carcinomas, hyperthyroidism, and angina without causing appreciable damage to contiguous structures.

Embryonic-type tissue and tissues exhibiting increased mitotic activity, such as certain neoplastic processes, tend to show a greater uptake of certain radioactive elements than the surrounding normal tissues. This permits definition and removal of the affected tissues and challenges research workers to develop methods of therapeutic irradiation using this principle.

A somewhat unique application of a similar technique has been applied to cells of the reticuloendothelial system. By in-

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The author thanks Drs. L. A. George and L. K. Bustad, Biology Operation, Hanford Laboratories, General Electric Co., Richland, Wash., for their assistance. Some of the clinical material was presented by Dr. Bustad in a paper, "Isotopes in Biology and Medicine," at the annual convention of the Norwegian Veterinary Medical Association in 1953.

travenously injecting radioactive isotopes in colloidal form, the Kupffer cells of the liver phagocytize a large percentage of the large particles, while smaller particles tend to be removed by other reticuloendothelial cells. If low energy *beta* ray emitters are used, the range of radiation therapy is extremely restricted.

The most common use of radioisotopes lies in the substitution of the radioactive element for the natural element in chemical compounds, thus "tagging" the element so that it can be traced by use of a sensitive monitoring instrument. Radioisotope tagging or tracer technique can be demonstrated in the case of penicillin. Why penicillin is more specific for some organisms than for others has been studied by culturing the penicillin with radiosulfur. After applying this radiopenicillin to susceptible organisms, it was found that penicillin was absorbed within the cell substance of the bacterium interfering with normal metabolism. Similarly, when isoniazid, a drug showing bacteriostatic effect on the tuberculosis bacillus, was labeled with radioactive carbon, it was shown that susceptible organisms incorporated the drug, whereas resistant organisms took up practically none of the compound. These experiments suggest that isoniazid and penicillin participate actively in the metabolism of the cell, thus blocking essential metabolic processes.

Cardiac function has been measured by injecting radiosodium into the vein of an extremity while a Geiger counter is placed over the heart region. As the radiosodium enters the chamber of the right side of the heart, the counting rate rises sharply for about one second and then drops back toward zero as the blood enters the lungs. The counting rate again rises, but less sharply and to a lesser extent, as the blood emerges from the lungs and enters the left side of the heart. The time lag between the two peak rates, and their relation to each other, give valuable information regarding heart function and circulation time through the heart. This procedure can be carried out quickly and provides data difficult to obtain by other means.

Blood vessel constrictions, atherosclerosis, and other circulation abnormalities have been studied and diagnosed by injecting radioactive saline solutions into the affected part and measuring its disappearance with sensitive counting instruments. Appendages with poor circulation exhibit a typically slow absorption curve, indicating to some extent the type of abnormality present.

Radioiron has been administered in the so-called "bone marrow" or "iron-utilization" test. A portion of the injected iron is incorporated in the developing red blood cells, making it possible to measure the amount of a given dose of iron that is utilized by the erythrocyte within a certain time interval. Such data afford criteria for normal and abnormal iron metabolism. Using this method, the life cycle of the erythrocyte can also be estimated. Red cells which have incorporated radioiron will continue to be radioactive during their lifetime, but new cells will not show activity after the blood serum level becomes negative.

Multiple injections of therapeutic quantities of radiogold in a colloidal form are being utilized in treatment of inoperable cancers. Radiogold has a tendency to become encapsulated in the form of "seeds" or sutures at the site of administration and, because of its short duration of activity or "half-life," it becomes less and less toxic to the immediately surrounding tissue. This tendency permits the administration of high-level initial doses without the possibility that large quantities of radiation will remain to cause further tissue damage.

The cause of gout has been investigated by means of nitrogen-labeled uric acid. Miscible pools of urates have been found to be up to 15 times larger in gouty human beings than in normal subjects. This suggests additional work in some fields of veterinary medicine, particularly in poultry-feeding research.

Radioactive gallium has been important in the study of bone metastases and bone-fracture healing. This isotope is an avid bone-seeker, especially concentrating in growing tissue such as the epiphyseal junction and in scar tissue resulting from injury. It is believed that radiogallium holds great promise in studies of bone healing and wounds affecting osteogenic tissue.

The tagging of certain insects with radioactive phosphorus has become common practice, and it is hoped that veterinarians will be able to apply this method with more of the parasites affecting domestic animals. What this application of radioisotopes can mean to the livestock

industry in the way of higher meat production is yet to be fully assessed, but it is expected that methods of combating these parasites will be vastly improved.

Use of I^{131} in determining basal metabolism may be taking the place of the oxygen-carbon dioxide balance method. By administering tracer doses of radioiodine to the subject and comparing the uptake of radioiodine by the thyroid gland with the excretion rate from the gland, an accurate thyroid function test is obtained. Further work must be done to see if this method can also be utilized in animal feedlots to correlate thyroid activity with weight gain. Determination of such a correlation would allow the breeder to save considerable money by eliminating unprofitable animals and being able to select his breeding stock earlier and more wisely.

DISCUSSION AND CONCLUSIONS

Despite some opinions to the contrary, the problems of the practitioner are uppermost in the minds of research veterinarians. They are trying to probe the cause of obscure sicknesses, the drug or therapy which will work best in a particular instance, and the reason it works best. We are closer to solving these problems today because of radiobiological techniques in research.

The veterinarian must undergo indoctrination in the field of radioisotopes. In the field of radiation biology, veterinary medicine is in the initial stages of knowledge and practice. Veterinary schools, especially departments of radiology and physiology, must take a new look at the whole subject of radioisotopes. If large programs of radiobiological research can not be attempted, then small scale experiments should be initiated. Our practitioners must become familiar with these problems if we are to succeed in this era of nuclear science. This is not an easy program. The Atomic Energy Commission has set up stringent regulations for the control of radioactive materials and for laboratory facilities where they may be used. Disposal of contaminated waste becomes a problem if large quantities of radioactive materials are utilized. Instrumentation is expensive and repair technicians are scarce.

Small scale experiments, however, may be carried out without elaborate facilities or instrumentation. The cost of isotopes is kept nominal so that institutions may take advantage of these tools in research.

The veterinary profession has been challenged to accept new duties and to learn new skills in a world committed to the use of increasing amounts of atomic energy. We must augment our technical training by the study and practice of radiation biology, and so master these latest tools of veterinary medicine.

The Veterinarian in Human Factors Research

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Today we are on the threshold of flight by nuclear-powered aircraft and, perhaps of greater significance, manned nuclear flight into space. However, before we can climb aboard, every detail of engineering and physiological response must be studied and tested, and measures must be taken to assure us that the air vehicle and its human cargo will return and be suitable for other journeys.

Perhaps the weakest link in this chain of knowledge and testing lies in the realm of human factors. The physical forces involved in rapid acceleration, deceleration, escape at high altitudes, and Mach numbers, as well as the psychological factors, must be solved if we are to operate modern aircraft effectively.

An additional stress encountered in nuclear flight is that imposed by radiation from the reactor which propels the plane. We must solve the problems of survival within a continuous radiation environment. In the case of a nuclear air base, this environment extends to ground personnel and, in the event of a crash, to even the civilian populace.

To understand these problems and to attempt to counteract the untoward effects of modern flight, the Surgeon General of the Air Force has a continuing program of research in its medical support activities. The veterinarian, as a member of the medical team, has been engaged in research projects in human factors.

From it's earliest inception, air force veterinarians have been associated with

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the nuclear aircraft program. A group of veterinarians on the staff of the School of Aviation Medicine have assisted in a study of the effects of irradiation upon the *Macaca mulatta* species of monkey. Military veterinarians at Oak Ridge (Tenn.) worked out many of the details of radiation damage in the burro, using both isotopic and atomic bomb irradiations. The burro resembles man in that his body size is similar and his life span is lengthy in comparison to most laboratory animals. Veterinarians at Hanford Atomic Products Operation have determined the hazards of radioiodine and other fission products associated with nuclear reactors in sheep and swine. These data can be partially extrapolated to man.

Some veterinarians may feel that these human factor problems of the Air Force are beyond the scope of veterinary medicine; that, since our specific training has been in the general area of animal health, we should leave all of this work to the physician. Unfortunately, there are but few experiments involving radiation toxicology which can be evaluated directly on the human being. As a result, all such work must be performed on animals similar to man—either primates which resemble man in many physical characteristics, large mammals which have a body size equivalent to or greater than man, or animals with long life spans, partially equal to that of man. For these studies, the veterinarian is the logical person to assume responsibility for experimentation. Who else is better qualified for this job?

The final clinical evaluation and extrapolations must be the result of teamwork between veterinarians, physicians, and other allied scientists. The veterinarian must accept the challenge of leadership in the field of animal experimentation which may ultimately benefit man. This position is his rightful place on the medical research team.

Artificial Light and Sexual Maturity in Chickens.—When chickens were raised in confinement and illuminated with various colored lights, there was no evidence of harm to sexual reproduction. While incandescent lamps gave best results, any quality of light stimulated onset of egg production three weeks earlier than in unlighted control pullets and, in males, it stimulated a significantly greater amount of sperm.—*Poult. Sci. (Jan., 1958): 102.*

Army Scientist Develops Anthrax Vaccine

The first nonliving anthrax vaccine acceptable for test in human beings was recently developed by scientists of the U.S. Army Biological Warfare Laboratories. The original vaccine, developed by Louis Pasteur in 1881 for cattle and sheep, and essentially the same vaccine used at the present time, can not be used in man.

In tests involving human beings, the new vaccine has been found to produce no toxic effects. Its effectiveness is being further evaluated by a cooperative project between the U.S. Government and Pennsylvania health departments by immunization of industrial workers in plants where anthrax is a hazard.—*Dept. of Army Res., Feb. 27, 1958.*

U.S. Cattle Inventory for 1958

Cattle numbers, on Jan. 1, 1958, were down 1 per cent from 1957 and about 3 per cent from their peak in 1956. This is only 6 per cent above the 1948 to 1957 average.

Slaughtering of cows was at a record rate in early 1957, much of it dairy stock. The milk cow and heifer total is the smallest since 1930.

Cow totals were far below their peak years in six major beef-raising states, especially in Texas and Kansas, and was only slightly higher in four states, including Mississippi and Alabama. Numbers of cattle on feed increased only in three cornbelt states and Florida.

The aggregate number of livestock and poultry was down 4 per cent from 1957 but improved prices provided a $3 billion gain in values. Beef cattle prices were up 30 per cent, milk cows up 20 per cent, hogs up 22 per cent, sheep up 30 per cent, and horses and mules were up 17 per cent.

The importation of 571,000 head of cattle from Canada and Mexico, during the first eleven months of 1957, was more than four times that in the similar period of 1956. The six states with the greatest cattle population were (in millions): Texas, 7.7; Iowa, 6.4; Nebraska, 4.7; Wisconsin, 4.3; and Kansas and Illinois, 4.0 each.—*Am. Cattle Producer (March, 1958): 9.*

Surgery and Obstetrics
and Problems of Breeding

The Surgical Treatment of Fractures of the Proximal Sesamoid Bones in the Horse

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FRACTURES of the proximal sesamoid bones have been observed in almost all classes of horses but most frequently in the racing Thoroughbred. A separation of the fractured fragments, with a weak fibrous union, often results. Recently, a description of surgical removal of fracture fragments of the proximal sesamoid was published.[1]

This paper describes two additional techniques for surgical removal of the fracture fragments. The first is the removal of fragments of the ventral portion of the bone and the second the successful treatment of old fractures complicated by a fibrous union.

The cause of such fractures is varied. Churchill[1] suggests that "fatigue which causes the horse to rely on basic skeletal structures rather than muscular activity" is one possible cause. Another cause which has not been sufficiently emphasized is the unequal tension applied to the sesamoid bones when the foot strikes the ground in an unbalanced position. We have observed several cases of sesamoid fracture which occurred while the horse was moving at a slow gallop. It was the opinion of the rider that fracture occurred as the foot of the animal hit a depression in the track or stepped on a stone.

We observed one Thoroughbred that had fractured a proximal sesamoid bone by stepping on a golf ball as he was walking across the track infield. Many times, sesamoid fractures of the forelimb are thought to be caused by a direct blow from the toe of the hindfoot. Such accidents seem possible but do not explain fractures occurring in the hindlimbs or bilateral fractures of the front legs. Rarely is there external evidence of skin trauma.

A high percentage of sesamoid fractures are at a right angle to the long axis of the limb. It is possible that this pattern is caused by the tension of the upper and lower sesamoidean ligaments. We examined one horse in which the lateral sesamoid was fractured and the medial one luxated dorsally. In another horse, both sesamoid bones were luxated dorsally about 1 inch. An instance has been described in which the sesamoid bones were broken in several places after the horse galloped in deep sand.[2]

The signs of a complete fracture are easily recognized. The horse may show little lameness until he starts to cool out after a race. At this time, extreme lameness, with swelling and pain of the fetlock joint, is noticed. In some cases, before swelling occurs, crepitation and separation of the fractured pieces of bone can be felt.

The prognosis is guarded or unfavorable as far as the horse's chances of successfully returning to racing are concerned. Fractures in which the parts do not separate may heal if the animal is given prolonged rest. Too often, the fractures heal with a fibrous union, only to separate again when the horse is put back into training. The fact that the sesamoid bone is only partially covered by periosteum and has no marrow cavity and little or no blood supply interferes with the normal healing process.

In some fractures, the bone fragments are in close apposition soon after occurrence but separate over a period of time (fig. 1, 2). This type of fracture can never heal properly with rest only.

In the past, the treatment of these fractures has been immobility with bandages or plaster cast. Removal of fracture fragments, as described,[1] gives encouragement to the surgical treatment of the condition. However, the author[1] stated that "in order to be a good surgical risk the fracture must involve the apex of the bone and the operation should be performed within two or three weeks of time of fracture." Our experience with the condition indicates that it is possible to operate successfully on fractures of several months' duration, and that it is also possible to operate on fractures involving the ventral portion, or base, of the proximal sesamoid bone.

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Case Reports

The following cases have been selected as examples of the various types of sesamoid fractures that we have encountered and include some that have been treated by surgery. All patients were placed on the operating table for surgery and given 1 oz. of chloral hydrate and 20 cc. of pentobarbital sodium intravenously. In addition, a bilateral volar nerve block was performed.

Case 1.—In the fall of 1955, this colt was was placed in training in preparation for racing as a 2-year-old. During an easy gallop, the colt suddenly faltered and pulled up lame, with a fracture of the proximal sesamoid bone. The colt was rested for six months but remained continually lame. On April 10, 1956, it was presented to us for treatment. Radiographs showed an old fracture with extensive exostosis of the right lateral proximal sesamoid (fig. 3).

The operation consisted of making an incision on the posterior surface of the sesamoid bone directly over the enlarged area, then locating and separating the fibrous union between the fragments of bone with a bone chisel. The roughened area was curretted until smooth (fig. 4). The

Fig. 1—Lateral radiograph of fetlock joint of a horse showing a fracture (arrow) of ventral portion, or base, of proximal sesamoid soon after occurrence.

Fig. 2—Same fracture shown in figure 1, two months later. Note how lower fragment has separated.

Fig. 3—Lateral radiograph of fetlock joint of colt (case 1) before removal of fragments. Notice extensive exostosis.

Fig. 4—Fetlock joint of colt (case 1) after removal of fracture fragments and exostosis.

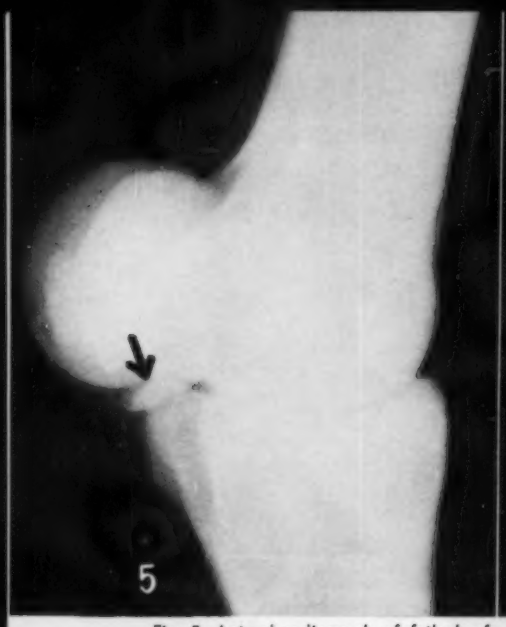

Fig. 5—Lateral radiograph of fetlock of gelding (case 2) two weeks after occurrence of the fracture (arrow).

Fig. 6—Fetlock joint of gelding (case 2) four months after removal of bone fragment.

incised tissues were sutured with No. 1 chromic catgut and the skin closed with Dermalon suture.

The colt was rested until the spring of 1957. When he was then placed in training, he showed no evidence of lameness from the sesamoid region; however, he developed "sore shins" and was blistered and rested for a month. Following this, he was again placed in training and has raced successfully, finishing in second place three times and in third place once.

Fig. 7—Lateral radiograph of fetlock joint of filly (case 3) taken immediately after fracture occurred. Notice the separated lower fragment (arrow).

Fig. 8—This radiograph was taken 14 months after fracture (case 3) occurred. Notice the apparent filling in of the fractured area and the resorption of the bone fragment.

Case 2.—In 1956, a 4-year-old Thoroughbred gelding with a fracture of the ventromedial portion of the right medial proximal sesamoid was treated (fig. 5, 6). The duration of fracture was approximately two weeks.

A longitudinal skin incision was made on the posterior aspect of the fetlock joint, at the point where the deep digital flexor tendon passes between the two sesamoid bones. With the fetlock joint slightly flexed, to relieve tension on the flexor tendons, an incision was made in the space between these tendons and the sesamoid bone, exposing the volar annular ligament.

The volar annular ligament of the fetlock was then incised to expose the deep flexor tendon and its sheath which was retracted to the side. This exposed the smooth fibrocartilaginous plate of the intersesamoid ligament in which the sesamoid bones are partially imbedded. An incision, at a right angle to the axis of the limb, was then made in the fibrocartilage over the fractured portion. By careful probing, the bone fragment was located, grasped with a pair of narrow-nosed forceps, and twisted or pried loose. The incision in the intersesamoid ligament, the deep flexor sheath, and the volar annular ligament were each sutured with No. 1 chromic catgut. The skin was closed with nylon sutures which were removed in ten days. This horse is now in training and has not shown any lameness.

Case 3.—On July 24, 1956, a 3-year-old Thoroughbred filly fractured the ventromedial portion of the medial proximal sesamoid bone of the right front leg (fig. 7). This filly was treated by putting the affected leg in a plaster cast for eight weeks, followed by supportive bandaging and stall rest for another two months. She did not show lameness when turned out to pasture. Subsequent radiographs of the fracture indicated that it was healing. However, when the filly was put back into training in September, 1957, she became lame almost immediately and continued so. She has since been retired to become a brood mare.

The fragment was not removed from this filly because the displacement was not great and subsequent radiographs (fig. 8) showed healing without any further displacement. The filly's inability to return to racing indicates that the fragment should have been surgically removed.

Case 4.—A 4-year-old Thoroughbred gelding fractured the right front lateral proximal sesamoid bone on Aug. 5, 1956. The colt was given ten months' rest and then put back in training. Lameness developed the first time he was worked for a half mile.

When presented for treatment 22 days later, a longitudinal incision was made directly over the enlarged area on the posterior aspect of the sesamoid area. It was found that the fractured portions pre-

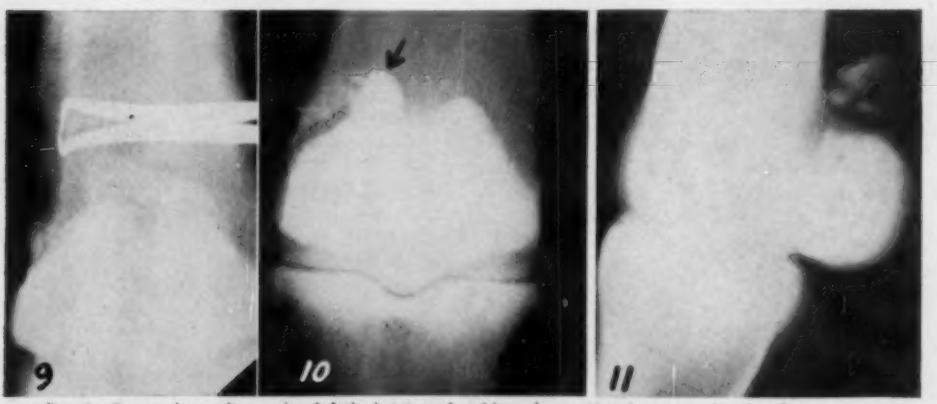

Fig. 9—Dorsovolar radiograph of fetlock joint of gelding (case 4) taken immediately after surgery. Small amount of exostosis still evident in soft tissues.

Fig. 10—This radiograph was taken one year following fracture of sesamoid (case 4). Notice the exostosis (arrow) and fibrous union of the fragments.

Fig. 11—Lateral radiograph of fetlock joint of gelding (case 5) taken prior to surgery, showing fragmented apex of sesamoid bone.

sented a fibrous union that was easily separated with a bone chisel. The roughened area was curretted to a smooth surface following removal of the fragments (fig. 9,10).

Within two weeks, this horse showed no signs of lameness while being walked one half mile twice a day.

Case 5.—A 3-year-old Thoroughbred gelding fractured the left lateral proximal sesamoid on the hindleg on Aug. 24, 1957. Nine days later, the fractured fragments were located at the apex of the bone and removed surgically.

This type of fracture is the most accessible and the simplest to treat. The fetlock joint capsule was incised where it pouches out between the suspensory ligament and the third metatarsal bone. The bone fragment (fig. 11) were then palpated and removed by grasping them with a pair of forceps and twisting them free, or by dissecting close to the bone fragment with sharp dissecting scissors. The joint capsule and skin were then closed.

GENERAL RECOMMENDATIONS

In sesamoid surgery, strict asepsis must be maintained at all times. It is important to suture all incised structures such as joint capsule, tendon sheaths, and ligaments. It is essential to be thoroughly familiar with the structures in the area to be invaded.

A solution containing 500,000 units of crystalline penicillin and 1 Gm. of streptomycin in 5 cc. of sterile distilled water is injected into the joint space when completing the closure. In addition, the horse is given daily doses of 3 million units of aqueous suspension penicillin, intramuscularly, for four days following surgery.

Aftercare of the leg has consisted of keeping the fetlock wrapped in sheet cotton covered by an elastic bandage and a muslin bandage to provide partial immobilization. The horse is kept confined in a box stall for a week, after which exercise is commenced. This consists of daily walking on grass or other soft surface. It is recommended that the horse be given four to six months' rest depending on the severity of the fracture.

Of the 12 horses treated for fracture of the sesamoid bones in this clinic, by surgical removal of the fragment, 3 have returned to successful racing, 3 are now in training, 3 are still resting (so the outcome is not yet known), 2 did not become sound, and 1 is undetermined.

CONCLUSIONS

Surgical treatment of fractures of the proximal sesamoid bones would seem to be indicated (1) in old fractures which had not allowed the horse to remain sound enough to train or race, and (2) in cases of recent fracture if the fragment is separated from the main portion of bone. When the fragment is in close apposition with the sesamoid bone, it is advisable to wait and see if union will take place.

References

[1]Churchill, E. A.: Surgical Removal of Fracture Fragments of the Proximal Sesamoid Bone. J.A.V.M.A., *128*, (1956): 581-582.

[2]Dollar, J. A. W.: Regional Veterinary Surgery and Operative Technique. Alexander Eger, Chicago (1912): 872.

Pneumothorax in Dogs

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Three dogs with pneumothorax were observed during the summer of 1957. This number was quite unusual for us and the cases are reported because they present a challenge in diagnosis.

CASE REPORTS

Case 1.—A male mixed Terrier, 2 years old, suddenly developed severe dyspnea and lassitude and stood with elbows abducted and head extended. The signs of distress were exaggerated when the dog was lying down. A diaphragmatic hernia was suspected and radiographs were taken.

A lateral view showed an area of air at the periphery of the thoracic cavity, and the apex of the heart was elevated away from the sternum. Another radiograph, taken after barium was given *per os*, showed the stomach and intestines to be in normal position.

Since the animal was showing so much distress, a diaphragmatic hernia with herniation of either the liver or spleen was suspected, even though it could not be de-

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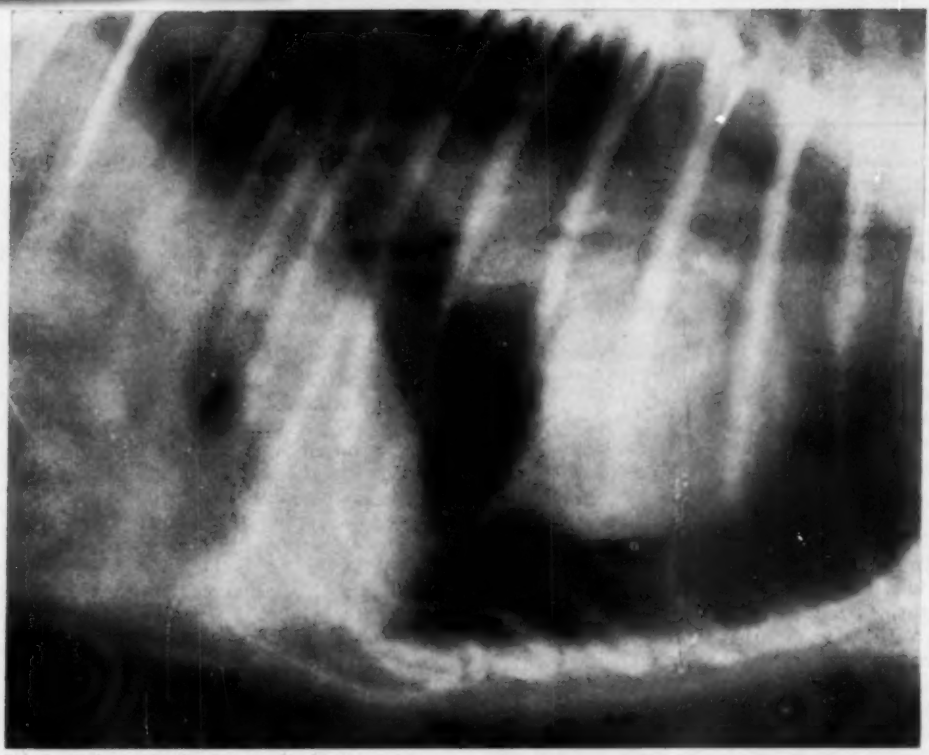

Fig. 1—Radiograph (lateral view), showing pneumonothorax in a dog. Notice the atelectatic lobes of the lung, the elevated position of the heart, and a zone of an accumulation of air in the pleural cavity surrounding the organs of the thorax. The diaphragm is intact.

tected radiographically. Thoracotomy was performed to see if the lesion could be seen and treated. A rent 5 cm. long and 0.5 cm. deep was found in the dorsal portion of the diaphragmatic lobe of the right lung. The damaged area was sealed with a large blood clot, so no repair was attempted, and the operative wound was closed. The dog, however, died without recovering from the anesthesia. The cause of injury was not determined.

Case 2.—A male Collie, 2 years old, suddenly developed signs of dyspnea, unrest, and anemia after being hit by a car. Radiographs showed a well-defined pneumothorax (fig. 1, 2). However, the dog died before it could be treated. At necropsy, a large volume of blood was found in the thoracic cavity.

Case 3.—A male Collie, 2 years old, suddenly showed signs of severe dyspnea and cyanosis. Radiographs showed this to be pneumothorax. There was no history of an injury.

In view of the outcome of the other 2 cases, it was decided to confine this dog im-

Fig. 2—Radiograph (ventrodorsal view) showing free air in the pleural cavity of a dog.

mediately to a cage equipped for oxygen. At the end of 12 hours, the improvement was sufficient so that no oxygen was needed and, at the end of four days, the dog's condition had improved sufficiently for him to be released.

CONCLUSION

These 3 cases of pneumothorax are reported because this number is unusual in our practice and because dogs showing these signs are usually victims of a ruptured diaphragm. Radiographic study is essential in the differential diagnosis.

Prolonged Gestation in Ayrshires.—Nine cases of prolonged gestation occurred in two years in a purebred herd of about 40 Ayrshire cows in Britain. The gestation periods varied from 336 to 381 days (56 to 101 days more than av.). Five of the calves apparently had died during parturition. The other 4 were destroyed within two days because of weakness. One could stand but showed tremors and a knuckling of the hind fetlocks. The birth weights (80 to 120 lb.) were greater than normal for the breed but less than if they had grown normally during the hypermature period.

At necropsy, the calves appeared normal but their hair was long and their hoofs and teeth were larger than normal. Two showed periorbital and submandibular edema.

At the normal calving time, the cows showed no relaxation of the pelvic ligaments but in a few cases the udders filled temporarily. At the time of parturition, the cows showed normal tissue relaxation and udder filling. Delivery was accomplished by fetotomy in one case and by considerable traction in two others. One cow died, most of them retained their fetal membranes, and 8 showed a postparturient lameness—a stringhalt-like action in a hindlimb for a few days. These findings were quite different from some others reported in the literature.—*A. L. Wilson and G. B. Young in Vet. Rec. (Jan. 25, 1958): 73.*

Treatment of Trichomoniasis in Bulls.—Of 25 bulls treated, in Poland, 19 recovered after one treatment, 5 with two treatments, and 1 bull was disposed of. The penis and prepuce were washed with warm water, dried, washed again with ether, then washed with 1 per cent solution of trypaflavine and smeared with 0.5 per cent trypaflavine ointment. The urethra was flushed with 50 ml. of 0.8 per cent trypaflavine.—*Vet. Bull. (Jan., 1958): Item 71.*

Estrus in Pregnant Cows

Estrus after conception was observed, in a 30-year period, at Washington State College, in 5.6 per cent of 6,751 pregnancies which resulted in normal calving in the Holstein-Friesian herd, and 55 per cent of these periods occurred within 35 days after conception. It occurred most frequently during the second and third pregnancies (7.5%). Twinning was 1.2 per cent higher in the cows with recurrent estrus than the herd's average, and the cystic tendency was considerably higher.—*J. Dai. Sci. (Feb., 1958): 267.*

X Rays and Spermatogenesis.—Spermatogonia are considered the most radiosensitive cells of the male germinal epithelium. They seem to be selectively damaged without impairing the other generations of developing spermatozoa.

In mice, exposure to 200 r resulted in a transient sterility about six weeks later. It is believed that five weeks are required for the testicular phase of spermatogenesis in the mouse, with an additional week for the epididymal phase. When male rats were given 500 r, either as a single dose or five daily exposures of 100 r, sterility developed in the seventh week and lasted for at least three months. Certain "radiomimetic" drugs may have a similar action.—*Nature (Feb. 1, 1958): 353.*

A Spermatocrit to Measure Spermatozoa Concentration.—A special centrifuge for estimating spermatozoa concentrations in bull semen is on trial. Centrifuging at 10,000 r.p.m. for ten minutes will pack the sperm firmly in the end of the tube (just as a hematocrit packs blood solids).—*J. Dai. Sci. (Feb., 1958): 318.*

A Missourian had 4 Montadale ewes (a new breed which resembles large Cheviots) which gave birth to a total of 14 lambs. All were doing well.—*Sheep Breeder (March, 1958): 15.*

A Diagnostic Test for Organic Phosphate Insecticidal Poisoning of Cattle

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DIAGNOSIS of anticholinesterase insecticidal poisoning of cattle by signs alone is difficult. In acute poisoning, the lesions found at necropsy are never outstanding and never pathognomonic.[8] Procedures[1,2] are available for the isolation and identification of parathion from animal tissues, but none for the organic phosphate insecticides as a group. A simple and rapid diagnostic test for these compounds would be an invaluable aid, not only to the practitioner, but to the laboratory toxicologist as well.

The mode of action of these poisons is concerned with cholinesterase activity in the nervous system. Although red blood cell cholinesterase activity is not in any known way concerned with nerve function, it does closely parallel the activity of nervous system cholinesterase. As such, it serves as an excellent, although not necessarily precise, indication of variation in the nerve cholinesterase activity. Also, inhibition of cholinesterase in both plasma and erythrocytes is the first demonstrable sign of parathion effect.[4] Since blood cholinesterase in cattle is limited to red blood cells,[9] a test on whole blood would seem applicable. In addition, for field work a test is required that would be relatively simple and rapid to perform. Such a test has been described[6] and modified[10] for the field testing of whole blood of human beings. The test depends upon the color change of bromothymol blue caused by the hydrolysis of acetylcholine iodide by acetylcholinesterase and subsequent increase in acidity.

At this laboratory it was found that, to obtain the same color development for the normal level of cholinesterase activity in bovine blood, a modification in the concentration of bromothymol blue, the volume of acetylcholine iodide, and the incubation period was necessary. The modified test, as used in this laboratory, is described.

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MATERIALS AND METHOD

The reagents and materials included: white blood cell (1:20) diluting pipettes; rubber-stoppered bottles to hold about 2 ml.; sterile, distilled water (pH 6.8-7.0); acetylcholine iodide solution (0.006 Gm. per ml.)—a quantity of powder sufficient for 10 ml. of solution may be conveniently weighed into a rubber-stoppered serum bottle and, before use, dissolved in sterile, distilled water; bromothymol blue (water soluble) (0.00025 Gm. per ml.) may be prepared in the same manner as the acetylcholine solution.

Shave an area over the veins on the upper side of an ear. Wash the skin with distilled water and then alcohol, and dry thoroughly with cotton. Puncture a vein with a clean, dry lancet. Draw blood up to the "5" mark on the pipette and wipe the outside of the pipette with gauze or tissue. Dilute the blood sample with the bromothymol blue solution up to the "11" mark. Shake the pipette briefly and deliver the contents into the bottle. Fill the pipette again to the "11" mark with the indicator solution and add to the solution in the bottle. Add one volume (to the "11" mark) of the acetylcholine iodide solution to the contents of the bottle. Rinse out the pipette several times with the contents of the bottle. Stopper the bottle, shake, and observe the initial color of the solution (it should be green). Observe the color by placing the bottle in a horizontal position above a white surface. Incubate the bottle at body temperature by placing it next to the skin or in a water bath at 98 F. for 45 minutes. At the end of the incubation period, the color of the solution is again observed. The following color changes have been reported[6] as representing the indicated percentage of normal cholinesterase activity in human blood: orange (100%); brown (75%); olive brown (50%); olive green (25%); and green (0%).

RESULTS AND DISCUSSION

Initial trials with cattle, using the original procedure[6] as adapted for human blood, were not satisfactory. After incubating the samples for the prescribed period of 20 minutes, there was an indicator change to olive green or olive brown instead of orange. The lack of correct color change probably resulted from the fact that the activity in bovine blood is confined to red cells and the activity in the cells is about one half that found in human cells. By increasing the length of the incubation period and decreasing the con-

TABLE 1.—Results of Blood Inhibited in Vitro with Parathion

Blood parathion (γ/ml.)	Bromothymol blue color change	
	Bovine blood	Human blood[10]*
0	Amber	Amber
5	Brown-orange	Amber
10	Brown	Brown
20	Olive brown	Olive brown
40	Olive green	Olive green
50	Green	.....
70	Green	.....
80	Green	Green

*Some variability of results shown in original table, but the majority of the samples gave the indicated color change.

centration of bromothymol blue and of the volume of acetylcholine iodide, the procedure described herein was developed. However, as in another study,[10] an amber color rather than an orange was found to indicate normal enzyme activity.

Blood obtained from healthy cows and calves with no known previous exposure to an organic phosphate insecticide was tested for cholinesterase activity. In every case, normal activity was found. To become acquainted with the various color changes due to enzyme inhibition, technical parathion in alcohol was added to blood samples, mixed, and allowed to stand at room temperature. The color changes (table 1) were essentially the same as found[10] for human blood subjected to a similar treatment.

To determine whether, in an animal poisoned with an organic phosphate insecticide, sufficient color change would occur to be valid for diagnostic purposes, 2 calves were given various levels of parathion orally. Dosages, color changes, and signs of poisoning are shown (table 2). In all three trials, a lowered blood cholinesterase activity was found. Although some time had elapsed between the administration of the insecticide and a detectable lowered enzyme activity, in trials 1 and 3, signs of organic phosphate insecticidal poisoning coincided with a definite color change. In trial 2, although a marked lowered cholinesterase activity occurred, only slight signs of poisoning were observed. This corresponds to the report[8] that daily small doses of a number of compounds progressively depressed cholinesterase to essentially zero with no objective symptoms being displayed.

The results of these trials show that, with described visible signs of poisoning and a marked color change indicating decreased cholinesterase activity, a diagnosis of poisoning is indicated. The results also indicate that the test has value in detecting suspected cases with no definite signs of poisoning. This is in accord with previous workers[5] who stated that cholinesterase measurements appear to be of value in confirming an exposure to anticholinesterase compounds, but offer little help in evaluating the degree of exposure.

Since the significance of low cholinesterase levels in blood is a controversial subject, because it is possible for an animal to appear normal with no detectable quantities of cholinesterase,[5] the test described herein should be used as a guide and only in conjunction with the signs and history. On the other hand, unequivocal inhibition of red cell cholinesterase must be demonstrated to justify a definite diagnosis, and

TABLE 2.—Test Color Changes and Signs of Poisoning in Calves Given Parathion

Days on test	Dosage (mg./kg.)	Bromothymol blue color change	Remarks
		Trial 1	
0	0	Amber	.....
1	10	Amber	.....
2	50	Amber	.....
3	75	Olive brown	Salivation, nervousness
4	.....	Olive brown	Signs of poisoning ceased
8	.....	Amber	Normal
		Trial 2	
0	0	Amber	.....
1	25	Amber	.....
2	25	Brown	.....
3	25	Olive brown	.....
4	25	Olive brown	.....
5	25	Olive green	Slight salivation, diarrhea Trial ended
		Trial 3*	
0	0	Amber	.....
1	50	Amber	.....
2	50	Olive green	Salivation, dypsnea, death

*Same animal as in trial 1 after a period of two weeks.

a diagnosis that is made in the absence of depressed cholinesterase levels is open to doubt.[4]

PRACTICAL USE

Human red blood cell cholinesterase has been reported[7] to be stable at storage temperatures ranging from 42 to 90 F. for six days. If the same is true for bovine blood cholinesterase, this would be an asset to the practitioner who is unable to perform the test. He then would be able to send samples to a laboratory. Oxylated blood samples from a normal and an experimentally poisoned animal were stored at room temperature and 38 F. for a period of seven days and tested daily for cholinesterase activity. The blood from the control animal gave a normal enzyme activity (amber color production) for seven days at both storage temperatures. However, a change was found in the color test for the blood of the experimentally poisoned animal. From the initial olive green, an orange brown was found after a storage period of 18 hours at both storage temperatures. Since it is generally concluded that cholinesterase inhibition by organic phosphate insecticides results from an irreversible phosphorylation of the enzyme, the explanation for the change is not known. It shows, however, that samples should be tested in the field or as soon as possible.

The advantages of the test as a diagnostic tool for cattle poisoned by the organic phosphate insecticides readily become evident. The small number and cost of reagents, the ease of preparation, and the inexpensive equipment make it readily adaptable to the practitioner. Compared to the manometric and electrometric methods, it is simple to perform. Of probably greater importance is that the test can be performed in the field and, consequently, the animals can be treated early.

SUMMARY

A simple and rapid colorimetric test for blood cholinesterase is described as an aid, in conjunction with signs and history, in the diagnosis of suspected or actual cases of cattle poisoned by organic phosphate insecticides.

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Septicemic Listeriosis in Lambs

Encephalitic listeriosis is probably the most frequent clinical form in sheep, but abortions also occur, as does sporadic septicemic listeriosis in newborn lambs.

In one flock, in Norway, 29 of 75 lambs became ill three to five days after birth. If not treated, they soon became prostrate, with an increased temperature and pulse rate.

All except 2 were given dihydrostreptomycin (250 mg.) and penicillin (300,000 units) parenterally. Although some were prostrate, they all recovered in 24 hours, usually after one treatment.

The 2 untreated lambs died in 48 hours. Their livers were crowded with whitish, necrotic nodules, and the spleens were swollen. Organisms cultured from the liver, spleen, and blood were identified as *Listeria monocytogenes*. The organisms were most sensitive to streptomycin, next to the tetracycline group, and penicillin was less effective. These organisms were able to survive at least three months in the feces.

A nutritional deficiency may have been a predisposing factor since the ewes had shown a marked tendency of "licking disease."—*O. Sandvik et al. in Nord. Vet.-med. (Jan., 1958): 17.*

The Therapeutic Use of Magnets Given Orally for Traumatic Gastritis

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IN EARLY 1955, after checking all available information on the use of magnets given orally for the control of "hardware disease," we finally obtained, at the insistence of a good client, a supply of magnets which were given to 50 of the adult cows in one herd. To date, there has been no further trouble, due to penetrating metal, in this herd which previously had many such cases. As far as can be determined, the cows have suffered no ill effects from the magnets themselves. We have, however, had little success in convincing clients of the value of using magnets prophylactically—probably not more than 200 animals so treated in two years—in spite of the fact that traumatic gastritis causes heavy losses in this area.

Most of our experience, therefore, has been in using magnets to treat animals showing signs of traumatic gastritis such as suddenly going off feed, stiffness in gait, sudden drop in milk production, pain on percussion under the sternum, grunting, slight elevation of temperature, and slowing or stasis of rumen movements. A few showed other less typical signs such as chronic or acute bloat, vomiting, and acetonuria. A majority of the animals had been sick two to four days. A few had acute flare-ups followed by periods of incomplete recovery for as long as six months. Magnets were not used in animals where splashing sounds or other evidence of pericarditis could be detected or in animals that were unable to rise or were moribund. Magnets, however, were given to all animals that seemed to have any chance of recovery.

Initial treatment consisted solely of the administration of a magnet plus an intramuscular injection of penicillin and streptomycin. In a few cases where indicated, a laxative was given 24 hours or more later.

Recovery occurred in 12 hours to three weeks in all but 9 of 83 cows so treated. Milk production was often restored to its original level, especially when the cows had not been milking long. Many of the recovered cows later calved without trouble and, to date, no recurrences in recovered animals have been reported to us.

Of the 9 cows which did not recover, 1 which died of pericarditis might have recovered if the owner had allowed us to give a magnet at the first sign of illness instead of four days later. Rumenotomy in another cow revealed an abscess on the floor of the reticulum but no metal was found in it. In this case, the magnet was returned to the reticulum and 90 days later the cow had recovered sufficiently in weight and condition to be sold for slaughter.

The other 7 of the 9 cows were slaughtered after failing to show improvement in ten days to two weeks. We were unable to follow these cases except to determine that none was condemned when slaughtered. In our opinion, there might be several reasons, other than misdiagnosis, why these 7 cows did not respond to the magnet treatment. Some might have recovered if given a little more time. Also, where the offending piece of metal is longer than the magnet, it will not be withdrawn and might even be driven deeper into the tissues by the weight of the magnet. In such instances, however, we believe that the magnet would prevent the wire from entirely leaving the reticulum and thus might prevent a fatality. Colleagues who have performed rumenotomies on cows which failed to respond to the magnet treatment have reported that magnets sometimes are found, not in the reticulum, but in the rumen where they are ineffective. We hope our newly purchased metal detector will help us discover this error in the future. It also seems probable that metal penetrating the dorsal part of the reticulum might be too high for the attraction of the magnet to be effective.

We believe, however, that the magnet is a more effective method of handling traumatisms than our previous method of giving symptomatic treatment on the first visit, followed by four days of starvation, confinement on a sloping floor, plus rumenatorics and antibiotic therapy. If that

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failed, we advised rumenotomy or immediate slaughter. The majority recovered under the initial treatment and a considerable number of the others recovered following rumenotomy; however, there were many recurrences and incomplete recoveries in both medically and surgically treated ones.

Because of the relatively small difference between the value of the cow for milking and for beef, in recent years, surgery or any expensive lengthy treatment has become unpopular with many dairymen, whereas the low-cost magnet treatment appeals to them; it affords hope of relief from the immediate illness and also of protection against recurrences. The "starvation treatment" was never popular with dairymen who like to feed their cows well, and many of them did not follow the treatment suggested. We have found that rumenatorics and laxatives are seldom indicated when magnets are used but, when indicated, the rumenatorics and laxatives seem to be more effective, probably because of the prompt relief from pain which seems to occur with the use of a magnet.

We have previously encountered considerable difficulty in handling "hardware disease" in cows nearing parturition. The starvation treatment was seldom effective due to the extreme fullness of the abdomen, while rumenotomy often led to serious complications such as abortion, retained fetal membranes, and metritis. Occasionally, if parturition occurred soon after the operation, the sutures would be torn out and the wound would reopen. The magnet has been a valuable aid in cases of late pregnancy.

Relief has been obtained in a few cows with chronic bloat and also in a few with signs of ketosis.

CONCLUSIONS

While time for observation and the number of cows treated have been too limited for definite conclusions, the results from using magnets therapeutically have been encouraging and an improvement over our previous method of treatment for traumatic gastritis. Some cattle which have carried magnets for two or more years have shown no ill effects. It is our hope that the apparent benefits from using magnets therapeutically will encourage our clients to have us use more of them prophylactically.

Multiple Hemoglobins in the Horse.—Finding of electrophoretically distinct hemoglobins in man has been followed, since 1955, by similar reports in cattle, sheep, and horses. Two hemoglobin fractions were found in all of 65 horses and there were strong indications of more than two in some. Only one fraction was found in 14 donkeys and it differed from the two in horses. Two fractions were identified in 19 mules and 3 hinnies, but there was evidence of the presence of three hemoglobins, one inherited from the donkey and two from the horse parent.—*Nature (Jan. 25, 1958): 267.*

Whirlpool Therapy for Horses.—A California racehorse trainer has devised a walk-in hydrotherapy unit for treating leg ailments of horses. It is made of steel and is 6 ft. long, 2½ ft. wide, and 4½ ft. high, with a water-tight door.

The water, heated to 108 F., is circulated at a rate of 150 gal. a minute and is 28 in. deep in order to cover the knees and hocks. A pound of Epsom salts and 1.5 lb. of natural mineral mud (colloidal bentonite) are mixed with each 3 gal. of water in an 800-gal. tank and the mixture is then pumped into the unit for 30-minute treatments. In addition to its therapeutic value, the treatment seems to have a relaxing effect.—*Blood Horse (March 1, 1958): 564.*

Prevention and Treatment of Cervical Abscesses in Swine.—On a farm where cervical abscesses in swine had been a recurring problem, 418 weanling pigs from fall and spring litters were self-fed a basal ration either alone (controls) or with chlortetracycline added at the rate of 50 or 100 Gm. per ton. There was no advantage in feeding the higher level (100 Gm.).

In the first two experiments, of the pigs which showed no signs of abscesses when treatment began, 29 of 53 control pigs (54%) developed abscesses as compared with 25 (21%) of 117 treated pigs. Results were almost as good when the 50-Gm. dose was reduced to 20 Gm. per ton from the time the pigs weighed 75 lb. until they reached market size. The treated pigs also showed increased gains and improved feed efficiency.—*H. E. Gouge et al. in J. Anim. Sci. (Feb., 1958): 34.*

A Simple Improved Method for Drawing Blood from Chickens

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CARDIAC PUNCTURE[1] is used for obtaining blood from chickens under 5 weeks of age. Their brachial veins are neither sufficiently large nor is the flow of blood great enough to permit samples to be made by venipuncture, as in older birds. Comparatively little difference was found[2] in the composition of blood samples from peripheral veins and the heart.

MATERIALS AND METHODS

In birds older than 5 weeks, an improved method of venipuncture is used in our investigations. It requires a 20-gauge hypodermic needle, 1½ inches long, and a 5-mm. glass syringe. The inside of the syringe and needle are thoroughly wetted with heparin solution (1,000 units of heparin per cc.).

The chicken is placed on its side, the legs bound with a wide elastic band around the metatarsal bones, and the wing nearest the table is folded around the upper wing so that the primary feathers of the former wing hold the ulna and radius of the latter (fig. 1). Under this type of restraint, the bird is immobile, leaving the operator's hands free, and the brachial vein of the wing nearest the operator is well exposed and tensed, due to extension of the scapulohumeral joint. Any feathers obscuring the brachial vein are plucked, and isopropyl alcohol is liberally swabbed on the site of venipuncture.

With a firm controlled thrust, the needle, bevel side up, is inserted into the vein; the brachial vein is held loosely by subcutaneous fascia, so sharp needles are necessary for this operation. Sufficient negative pressure is exerted in the syringe to draw the blood into it as quickly as possible without collapsing the vein. Brachial venipuncture does not seem to affect the bird and comparatively little resistance is encountered.

Immediately after the blood is drawn, the needle is removed from the syringe and, by gentle pressure on the plunger, the

Fig. 1—Photograph showing a simple type of restraint used in drawing blood from a chicken.

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blood is squeezed onto the inside of a collecting tube. For most studies, 5 ml. is routinely drawn.

DISCUSSION

The subcutaneous hemorrhage from a well-made venipuncture usually can be controlled by pressure with a small cotton pledget. If the area has not returned to normal in time for the next bleeding, the opposite side of the bird can be used.

In this investigation, heparin, sodium citrate, and sodium oxalate solutions were tried as anticoagulants. When the latter two were prepared according to the Wintrobe method[4] and dried in collecting tubes, they were inefficient in preventing cellular clumping. Heparin in an isotonic solution, therefore, was used almost exclusively, 1 part to 24 parts of blood. Heparin mixes poorly with blood,[4] and to overcome this difficulty, the tubes were inverted ten times immediately after the blood was placed in them. Since shaking increases thrombocytic and leukocytic clumping,[1] this mixing procedure must be done gently.

This method of drawing blood proved satisfactory in a study involving over 500 birds. It was particularly efficient for one operator working alone. The technique was used for drawing blood from birds on the range, in poultry houses, and in the laboratory.

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Lymphomatosis Virus in Saliva and Feces of Chickens

The virus of visceral lymphomatosis was found to be present in the saliva and feces of chickens when oral washings, fecal, and other extracts were inoculated into day-old chicks of a highly susceptible line.

When the chicks were inoculated at 1 day of age, the oral washings taken from them, at periods from 10 to 180 days of age, all contained infectious levels of the viruses of visceral lymphomatosis and of osteopetrosis.

Similar tests on noninoculated chicks, in contact with those inoculated, showed the saliva to be highly infectious at 90 and 180 days.

In tests on 5 apparently normal hens, 2 shed infectious levels of the virus in their saliva and feces. The infection in the chicks usually remains inapparent for a long period and may never result in overt disease.—*B. R. Burmester in Poult. Sci. (Sept., 1956): 1089.*

Hemorrhagic Enteritis of Turkeys

Hemorrhagic enteritis has been observed frequently in turkeys in the field since it was first reported in 1937, in Minnesota. It is distinct from the hemorrhagic syndrome in chickens.

This disease occurred at the Ohio Agricultural Experiment Station in flocks of 1,100 turkeys, first in November, 1955 (lasting 21 days, with a mortality of 1.6%); and again in January, 1957 (lasting 21 days, with a mortality of 3.5%).

An occasional bird was drowsy and showed bloody droppings, but the birds were usually found dead and, upon necropsy, showed little except anemia of the breast and thigh muscles, congestion of the mesenteric vessels, congestion and edema of the intestines (especially the duodenum), and a lumen full of sanguineous material.

No parasitic, bacterial, or viral agents could be implicated. The cause is unknown. —*Poult. Sci. (Nov., 1957): 1267.*

Furazolidone and Ornithosis.—When 125 young poults were inoculated with ornithosis virus and immediately given an all-mash feed containing levels of 100 to 300 Gm. of furazolidone per ton, for two weeks, it apparently had no prophylactic value against the disease.—*D. E. Davis et al. in Avian Dis. (Feb., 1958): 117.*

Cooling Time and Toughness in Fryers. —The relative tenderness in chicken fryers is directly associated with the aging period between slaughter and freezing. Those held 24 hours were the most tender, but for 10-week-old fryers, three to six hours of cooling time was sufficient.—*Poult. Sci. (Jan., 1958): 231.*

A Suspected Case of "Hard Pad" Disease in a Dog

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"Hard pad" disease of dogs, which was first described in England and is characterized by a course similar to canine distemper, has never been reported before in Korea, as far as we know. It is generally believed that canine distemper and hard pad disease of dogs are separate entities.[1,2]

The purpose of this paper is to describe a case which was highly suggestive of hard pad disease.

Case History and Clinical Features

A mongrel female dog, 6 months old, had been sick more than 40 days. She was treated at a small animal clinic but became apathetic, lost her appetite, and had a high fever, diarrhea, and mucopurulent discharges from the eyes and nose.

The patient could scarcely stand and showed a staggering gait when forced to walk. There was marked depression, emaciation, and anorexia. Her sleepiness was partly due to phenobarbital medication which had been used to relieve the severe nervous disturbances. The pupils were dilated and there was no reflection. The left cornea was slightly cloudy in the central portion. The visible mucous membrane and conjunctivas were pale and anemic. There was a high rectal temperature (39.8 C.), and mucopurulent discharges from the nose and eyes were profuse.

Skin eruptions on the abdominal area and hyperkeratosis on all foot pads[1,2] (fig. 1) were noticed. Signs of severe diarrhea were also observed.

Because of the pneumonic signs and the advanced condition of the case, further treatment was not given.

Postmortem Observations

The principal lesions found at necropsy were: a small ulcer on the left cornea; pustular eruptions on the abdomen; and mucopurulent discharges from the nose and eyes. The liver was moderately edematous, swollen, and congested. Petechiae were present in the cortex of the kidneys.

Fig. 1—Hyperkeratosis of the foot pad of a dog.

The alimentary tract showed petechiae and slight inflammation.

The lymph nodes, especially the abdominal nodes, were edematous. There were myocardial degeneration and excessive fluid in the pericardium. Bronchial pneumonia was marked in the right lung. There was slight congestion of the meninges and brain proper.

Histopathological Findings.—The micro-

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The author thanks Dr. Chang H. Rim for his technical assistance in preparing the section preparations.

scopic examination was mainly directed toward the detection of the specific inclusion bodies in the various tissues, such as urinary bladder, liver, ependyma, cerebrum, and cerebellum. No inclusion bodies were found in the preparations.

DISCUSSION

Since neither immunological tests nor virus identification were achieved in this study, nothing can be said with certainty as to the etiology. However, since there was a specific digital hyperkeratosis, with clinical signs similar to those of canine distemper, and as there were no specific inclusion bodies which are suggestive of either canine distemper or infectious canine hepatitis, this may have been a case of hard pad disease.

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Ocular Manifestations of Toxoplasmosis in Man.—Acquired toxoplasmosis may vary from a mild, one-day, febrile illness to a fatal, widespread disseminated infection. In adults, it is rarely recognized unless confirmed by serological tests.

Congenital toxoplasmosis occurs when the pregnant woman acquires infection, usually inapparent, which is transmitted placentally to the fetus, often causing irreparable damage to the central nervous system.

In man, the most important manifestation is chorioretinopathy in the congenital form, and focal choroiditis in adults.—*J.Am.M.A. (Jan. 18, 1958): 299.*

Agammaglobulinemia in Man.—Agammaglobulinemia, a condition in which the patient suffers from repeated bacterial infection, occurs in three forms. They are: (1) congenital agammaglobulinemia which affects only males and, like hemophilia, is transmitted from mother to son with half of the sons being affected; (2) acquired agammaglobulinemia which occurs in both men and women who seemed normal until they became adolescents or adults—there may be an inherited tendency toward this condition as in diabetes; and (3) temporary agammaglobulinemia, which occurs in babies and may be the most common form.

The *gamma* globulin in the blood of a newborn child is acquired from its mother and normally the child begins to make its own when 4 to 12 weeks old. However, if this is delayed, the child may suffer from severe bacterial infection unless given injections of *gamma* globulin.—*J.Am.M.A. (Feb. 1, 1958): 481.*

Female Hemophiliacs.—An unusual transmission of the hemophilic gene in a family in which only males had been previously affected is reported. When a woman carrier married her hemophilic first cousin, their three sons were normal but two of their three daughters suffered from hemophilia. Both affected women married men with no hemophiliac history and each couple had one daughter, both of whom had hemophilia.—*Brit. Med. J. (Dec. 28, 1957): 1528.*

Relaxin.—Relaxin, which was first discovered in the serum of pregnant rabbits in 1926, when it was found to relax the pubic symphysis when injected into virgin guinea pigs, has been prepared from an extract of the ovaries of pregnant sows and has also been isolated from rabbit placentas. Not only does it relax pelvic connective tissues but it increases the elasticity of the skin. It has been used in cases of scleroderma in man for periods up to 30 months without undesirable side effects.—*J.Am.M.A. (Jan. 25, 1958): 319.*

Quick Test for Albuminuria.—A new tablet test for albuminuria was found to be rapid, convenient, and accurate, except for trace amounts of albumin. One drop of urine is placed on the tablet followed, when it is absorbed, by two drops of tap water. In ten seconds, the color (yellow to deep blue) is compared with the provided chart which indicates the albumin content of the urine.—*J.Am.M.A. (Nov. 9, 1957): 1277.*

Statistics on Ailments of Cats

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A few weeks ago, a questionnaire from the American Animal Hospital Association inquiring about cats stimulated us to survey our feline practice. In our practice, we handle about 5,000 animals per year, of which 700 (14%) are cats. The tabulation (table 1) covers 2,045 entries over a three-year period (Jan., 1955, to Nov., 1957), and the cases are classified into nine general divisions and further into more specific subdivisions. The outcome of each case is also indicated.

The results, as tabulated, contain a minor element of error in both the "recovered" and "died" columns. For example, when an animal is designated as recovered, it usually means that it was well or greatly improved; but if there was a relapse, it may have died or have been euthanatized elsewhere without our knowledge.

Conversely, the deaths include some animals that would have recovered but were euthanatized because of lack of funds or lack of interest on the part of the owner (see trauma). However, it is our guess that the errors in the two columns approximately balance each other.

It is of interest that the conditions tabulated under the urogenital system were the most numerous (560). Castration of the male was the operation most frequently performed (195), with spaying (182) a close second. The subcutaneous abscess was the ailment most frequently treated (193). Since abscesses are usually the result of fight wounds and since castrations, both male and female, tend to reduce the incidence of fighting, it is significant that these three figures account for approximately 25 per cent of the total cases tabu-

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TABLE I—Number of Recoveries and Deaths in 2,045 Cats Treated Over a Three-Year Period

Nature or location of the ailment	Cases	Recovered	Died
1. Urogenital:			
Castration, female	182	181	1
Castration, male	195	195	0
Dystocia	25	21	4
Pyometra	11	8	3
Complications of pregnancy	19	14	5
Cystitis and urine retention	88	68	20
Chronic nephritis	40	15	25
Total	560	502	58
2. Infectious:			
Panleukopenia	143	100	43
Infectious anemia	33	17	16
Pneumonitis	211	200	11
Fever, ideopathic	21	20	1
Central nervous disturbance	27	16	11
Respiratory	34	31	3
Pyothorax	4	0	4
Total	473	384	89
3. Gastrointestinal:			
Digestive, miscellaneous	32	30	2
Poisoning, known or suspected	5	2	3
Impactions	13	8	5
Pancreatitis	2	0	2
Jaundice, noninfectious	2	1	1
Fish-eaters syndrome	12	8	4
Foreign body	2	2	0
Total	68	51	17
4. Trauma:			
Injuries, general	61	48	13
Fractures	70	41	29
Luxations	7	7	0
Diaphragmatic hernia	2	0	2
Abscesses	193	188	5
Osteomyelitis	23	21	2
Total	356	305	51
5. Skin:			
Dermatitis	99	96	3
Cutaneous granuloma	18	18	0
Labial granuloma	5	4	1
Alopecia	12	12	0
Pediculosis	1	1	0
Otitis, parasitic	26	26	0
Onychosis	1	1	0
Total	162	158	4
6. Oral cavity:			
Foreign body	8	7	1
Stomatitis	21	19	2
Dentistry	41	41	0
Total	70	67	3
7. Neoplasia:			
Types not classified	26	6	18
Total	26	6	18
8. Heart and vessels:			
Acquired	9	3	6
Congenital	1	1	0
Total	10	4	6
9. Miscellaneous:			
Vaccination - panleukopenia	36	36	0
Vaccination - pneumonitis	24	24	0
Vaccination - rabies	2	2	0
Onychectomy	5	5	0
Osteogenesis imperfecta	6	2	4
Unclassified	64	55	9
Board	143	143	0
Euthanasia - not treated	40	0	40
Total	320	267	53

lated. Heavy traffic was the most serious hazard to cats in downtown Buffalo, accidents accounting for 140 entries or 7 per cent of the total.

Panleukopenia was the greatest single cause of death from an acute illness (43), and diseases of the urinary tract accounted for the largest number of deaths from chronic illness (nephritis 25, cystitis and retention 20). Malignant tumors, comparatively rare in the cat (26 cases), had the highest percentage of mortality (69%).

Statistically, the response to treatment of skin ailments looks good, but recovery was slow and recurrences were common although not fatal.

Tetanus in a Cat.—A mature male cat died of tetanus, in Ceylon, three days after it was noticed to be stiff. There was a deep wound in one limb, pronounced reflex excitability, the pupils were contracted, and the nictitating membranes were prominent. Records were found of only 5 previous cases in cats. The cat has been estimated to be 2,400 times more resistant to tetanus than the horse.—*Brit. Vet. J. (Feb., 1958): 48.*

Mosquito Bite and Allergy.—Before showing a reaction to mosquito bites, rabbits had to be sensitized by ten to 12 previous bites. When experimentally bitten twice a day, the peak sensitivity occurred on the thirteenth day regardless of the species of mosquitoes used.—*Sci. News Letter (March 8, 1958): 149.*

To Compare Hog Cholera Vaccination Methods.—The California Advisory Committee on hog cholera is requesting a delay in the enforcement of a congressional bill to ban the use of live virus in hog cholera vaccination. They are asking the School of Veterinary Medicine, University of California, to conduct a trial comparing the use of modified virus as against the live virus for the control of cholera in garbage-fed hogs.—*California Farmer (March 15, 1958): 279.*

An Iowa Embargo on Feeder Pigs.—A temporary ban has been placed on shipment of feeder pigs into Iowa because of increasing incidence of hog cholera and lung infection in pigs, chiefly from southern states. About 600,000 such pigs are imported annually. Fifty to 75 per cent of some groups of these pigs have died within a few days after arrival.

The state requires that pigs be given anti-hog cholera serum and live virus vaccine, either 30 days before shipment or immediately after arrival, and be accompanied by a health certificate from a veterinarian at the point of origin. Many of these pigs have internal parasites which are found only in the deep south.—*Nat. Hog Farmer (March, 1958): 30.*

Effect of Chlorinated Naphthalene on Poults

Since chlorinated naphthalene was found to be a cause of hyperkeratosis in cattle, its possible toxic effect on poultry has been studied.

Chlordane, a chlorinated hydrocarbon structurally similar to chlorinated naphthalene, was reported, in 1954, to be toxic to poults, whereas lindane was nontoxic. Aldrin, a chlorinated naphthalene derivative, when fed to turkeys at levels of 12.5 and 25.0 p.p.m., caused mortalities of 15 and 100 per cent, respectively.

In recent trials with three chlorinated naphthalenes, a mixture of penta- and hexachloronaphthalenes (Halowax 1014) caused high mortality and depression in gain of poults while the more highly chlorinated octachloronaphthalene (Halowax 1051) had no apparent adverse effect, possibly because of its high melting point and low solubility.—*Poult. Sci. (Jan., 1958): 185.*

Growth Under Different Environmental Conditions.—The comparision of 898 American-born Japanese children, in California, with similar children in Japan showed the former to be significantly taller, heavier, and more advanced in skeletal development at every age from 6 to 19 years for boys, and 6 to 14 years for girls. The difference was much greater than anticipated. This indicates a need for caution in interpreting relatively retarded growth under less favored conditions as basic genetic differences.—*Science (March 7, 1958): 515.*

A new world's record price of $17,000 was recently paid for a Polled Hereford female in Florida.—*The Cattleman (Feb., 1958): 70.*

Tetany in Cattle on Winter Rations. Part I— A Clinical Report

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HYPOMAGNESEMIC TETANY has usually been reported either soon after pasturing cattle on young lush growth,[12,14,17,20] especially where green wheat, rye grass, or other cover crop is involved,[15,16] or in cattle on pasture during the winter.[13,21] A few cases have been reported to be the result of unbalanced and magnesium-deficient rations or underfeeding.[6,19]

For ten years, beef cattle with signs of tetany have been observed in the Allegheny Plateau area of extreme western Maryland and also in adjacent areas of West Virginia. With rare exceptions, tetany in this area is seen after pasture growth has stopped. The incidence steadily increases from January to an April peak, then rapidly declines after pasture reappears. Many cases develop during the first three weeks after parturition. Rarely, cows may be affected immediately before parturition or as late as five months after. Tetany has been diagnosed in a limited number of calves and nonlactating cows. No affected adult males or steers have been observed. The incidence increases with age and there is a tendency to recurrence in some individuals. A few herds account for a high percentage of cases, but many small and scattered herds have suffered losses.

TETANY SYNDROME

Affected cows often display incoordination progressing into paresis and coma. Some cows are alert and apparently normal but are unable to rise although they make repeated and valiant efforts. The rectal temperature, except when taken after struggling, is usually slightly low. At the onset, a cow may leave the herd and may show a stiff, high-stepping gait, muscular tremors, staring eyes, an "anxious" expression, and variable inappetence. Evidence of poor vision, twitching ears, violent blinking, grinding of the teeth, foaming at the mouth, bloat, and staggering may be noticed. Cows showing signs of tension, fright, or irritability occasionally may attack attendants.

The acuteness of the tetany syndrome may also vary widely. Cows have been observed to fall and die immediately after such normal activity as walking or eating; however, sudden attacks usually follow excitement or running. Cows with a high degree of irritability often fall in convulsions or, if down, may be precipitated into convulsions by noise, movement, or preparations for treatment. Tetany incidence apparently is not limited by breed but Aberdeen Angus cattle, which are less common in the area, are not represented by serum samples. When diagnosed in Aberdeen Angus cows, the course of tetany has tended to be more acute and more often fatal than in the average cow.

Many cases may formerly have been misdiagnosed as poisoning. The variability of the syndrome hampered efforts at diagnosis, but eventually a pattern suggestive of grass tetany was perceived. In April, 1955, serum samples from 9 cows with representative cases were analyzed* (table 1) using the Clark-Collip modification of the Kramer-Tisdall method for serum calcium determination, and the method of Denis, modified, for serum magnesium determinations.[10]

In the tetany season of 1955 there were 42 cases, 15 of which required re-treatment. Only 26 cases and nine re-treatments were recorded in 1956; no serum samples were taken. In 1957, by the end of February there had been 28 cases, 9 of them retreated. The 1957 season totals were 107 cases and 22 relapses. During March and April, 1957, case histories and serum samples were collected from 11 cows with diverse types of cases. The interval since

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*Determinations were made by Professor Robert B. Johnson, of the Maryland Live Stock Sanitary Service Laboratory, College Park.

TABLE 1—Comparison of Serum Calcium, Calcium to Magnesium Ratios, and Magnesium Values in Cows, in 1955

Case No.	History	Serum Ca, (mg./100 ml.)	Ca-Mg ratio	Serum Mg, (mg./100 ml.)
1	Comatose; re-treated two or three times.	4.6	more than 46.0-1.0	less than 0.1
2	Mixed breed;* parturient paresis appearance.	4.7	more than 4.7-1.0	less than 1.0
3	Excited, fell, convulsed; treated, fast recovery.	5.1	more than 5.1-1.0	less than 1.0
4	Down, later re-treated; finally recovered.	5.2	more than 5.2-1.0	less than 1.0
5	Nervous but on feet.	5.4	6.8-1.0	0.8
6	Staggering typically.	5.8	5.3-1.0	1.1
7	Very nervous, on feet.	6.1	7.6-1.0	0.8
8	Typically nervous.	7.0	11.6-1.0	0.6
9	Very nervous, on feet.	7.1	17.1-1.0	0.4

*All other cows were Herefords.

calving and other details are summarized (table 2).

TREATMENT

Before the described condition had been differentiated from nonspecific poisoning, some recoveries were observed following oral administration of a proprietary remedy containing magnesium sulfate (Scarlet Drench**). Later it was discovered that intravenous infusion of one of the commercial solutions containing calcium, magnesium, and dextrose (D.C.M. Solution, Special Concentration**) was much more effective. In 1957, severe cases usually were given, intravenously, 500 ml. of a solution containing calcium, magnesium, dextrose, and phosphate (Norcalciphos**). Part of a 500-ml. dose of a similar, less concentrated preparation (Parcal**) was often used intravenously in less acute cases. The remaining solution was well distributed subcutaneously over several areas for slower absorption. To avoid excitement from additional restraint and to hasten administration, the milder solution was occasionally given by the intraperitoneal route to nervous, standing cows. A few cows treated while undergoing convulsions were given supplemental doses of chloral hydrate and magnesium sulfate solution (Mag-Choral relaxant**) to effect.

PROGNOSIS

Prognosis is based on the appearance of the animal a few minutes after treatment.

**Scarlet Drench and Mag-Chloral relaxant are produced by Haver-Lockhart Laboratories, Kansas City, Mo.; D.C.M. solution, by Jensen-Salsbery Laboratories, Kansas City, Mo.; Norcalciphos, by Norden Laboratories, Lincoln, Neb.; and Parcal, by Pitman-Moore Co., Indianapolis, Ind.

TABLE 2—Comparison of Serum Calcium, Calcium to Magnesium Ratios, and Serum Magnesium in Cows, in 1957

Case No.	Interval since parturition	Description	Serum Ca, (mg./100 ml.)	Ca-Mg ratio	Serum Mg, (mg./100 ml.)
10	1 wk.	Agitated, unsteady, fell; alert.	4.2	more than 42.0-1.0	less than 0.1
11	4 wk.	Periodic inappetence; weak; comatose.	4.3	6.4-1.0	0.7
12	3 days	Grinding teeth; staggering; flat.	5.2	more than 52.0-1.0	less than 0.1
13	6 wk.	Unsteady, fell, struggled; weak.	5.5	9.2-1.0	0.6
14	4 mo.	Down, treated before; inappetence; reeling.	5.7	19.0-1.0	0.3
15	5 wk.	Stiff, staggering; flat; re-treated.	6.0	4.6-1.0	1.3
16	2 mo.	Convulsion; recovery; appeared normal.	7.8	12.9-1.0	0.6
17	12 days	Inappetence; nervous; excited, fell.	8.6	17.1-1.0	0.5
18	3 mo.	Inappetence; dazed; unsteady; died.	8.6	12.2-1.0	0.7
19	10 days	Bright, eating; posterior paresis.	9.8	8.2-1.0	1.2
20	1 wk.	Re-treated; bloat; foul diarrhea.	9.1	5.3-1.0	1.7

If the cow is then able to rise, recovery is usual. The chance of recovery decreases as the interval since onset, or the degree of motor irritation, increases. Cows which required re-treatment after failure to rise in a few hours rarely recovered unless the improvement during initial treatment was marked. Relapses sometimes occurred in a few hours; in other cases, they were delayed for several weeks. Although records are not available, it is estimated that roughly 80 per cent of the affected cows recovered with one treatment and about 10 per cent ultimately died, usually following re-treatment or during a relapse. About 1 per cent of the cows failed to respond to any treatment.

CASE HISTORIES

The 20† cows sampled were located on 15 different farms.

Case 10.—A Shorthorn cow, 8 years old, was seen apart from the herd and agitatedly lowing for her calf. When driven toward the nearby barn, she fell and was unable to rise. After half an hour, she was able to rise and enter the barn. When examined, she had been down for three

†Information on cases 1 to 9 is reported in table 1.

hours; her temperature was 101.4 F. She got up with help 20 minutes after treatment.

Case 11.—Periodic inappetence had been observed for two weeks in an Angus-Holstein cow, 8 years old. She had not eaten for 24 hours, was weak in the morning, and was down at noon. When seen at 5 p.m., she lay doubled in a corner and was unable to stand up when urged. Her temperature was 100.3 F. and her urine showed +2 albumen. She responded slowly to treatment but was up four hours later.

Case 12.—At 3:00 p.m. this 9-year-old Shorthorn was staggering, grinding her teeth, and foaming at the mouth. She was down at 5:30, flat on her side by 8:30. Her respiration was rapid and shallow, and the mandibular pulse was weak as treatment was begun. The pulse became strong after 175 ml. of infusion, then weakened, and was indistinguishable after 250 ml. had been given. The cow got up ten minutes after receiving 500 ml.

Case 13.—This 6-year-old Shorthorn walked unsteadily in the evening. The next morning she was down and her neck chain was tight around her neck, but she was able to rise when freed. When examined at 4 p.m., she was down and quiet but had head and hip bruises from earlier struggles. Her pulse was initially strengthened but became very weak during infusion of the final 125 ml. of solution. She was not quite able to rise with help but she was on her feet the next morning.

Case 14.—This Hereford, 10 years old, had been thin since parturition. She had been down and treated for tetany three days previously and had eaten progressively less since then. She was able to rise and stagger around but had a weak pulse and a temperature of 101.2 F. Samples of her urine had a musty odor and a trace of albumen. The owner observed no response to treatment and later killed her.

Case 15.—This Hereford had been walking stiffly for three days, but seemed normal otherwise. At 8:00 a.m. she staggered and was glassy-eyed; at 9:00 a.m. she was down on her side. When examined at 9:30, her temperature was 102.0 F. and yellow crusts were noticed around the reddened eyes. A nasal discharge was present. She made a poor response to treatment, was retreated, and is believed to have died later.

Case 16.—This 6-year-old Shorthorn was observed in tetanic convulsions. Within 30 minutes she had spontaneously recovered and had walked to the barn. When seen, she was standing normally, eating hay. The owner had lost several cows after similar attacks over a period of years.

Case 17.—The retained fetal membranes were removed from this 4-year-old Hereford ten days before this attack. She had eaten less than normal for two days, and for several hours had been reeling slightly and making chewing movements. When caught for treatment, she went down and was unable to rise. She had an acetonuria of +1. The cow was able to stand five minutes after treatment.

Case 18.—Since calving two months previously, this 8-year-old Hereford had been losing weight. She had eaten poorly for ten days, and did not give enough milk to satisfy her calf. Her movements were unsteady and she appeared dazed. Her temperature was 101.2 F. Treatment and re-treatment were ineffectual. She died two months later, and fat necrosis was found in the renal area. The results of a necropsy of another cow with a similar history, later on the same farm, were grossly negative.

Case 19.—This Hereford, 7 years old, had difficulty in rising. Before treatment, she was bright and ate and defecated normally. She was able to rise on her forelegs, but her hindlegs were weak. Ten minutes after treatment she was able to rise and walk.

Case 20.—On April 10, this cow calved and was treated for tetany. Four days later, she was apparently normal except for bloat that was relieved by tetany treatment. On the seventh day she was again bloated, had a malodorous diarrhea, and a temperature of 102.3 F. Tetany treatment, given after taking blood, again relieved the bloat and the cow was promptly sold.

DISCUSSION

These case histories are derived from notes submitted to the laboratory with the serum samples. Accordingly, they report deviations from the usual, but often fail to compare specific details. Also, some observations, such as on urinalysis and on variations in pulse rate and respiratory rate, were not regularly made. The variations in consistency of feces were not mentioned.

The mention of albuminuria in cows 11 and 14 is of interest, in view of the state-

ment that albuminuria is regularly found in grass tetany but not in parturient paresis.[17] In New Zealand, acetonemia often accompanies tetany,[18] as in cow 17. In contrast with the findings in cow 18, necropsies of other cows suspected of having tetany showed results which were uniformly negative. The absence of specific lesions is reported in cows with tetany in England.[1] Some of the exceptional signs in case 15 may possibly be related to leptospirosis then occurring on that farm.

From the serum values found in 1955 (table 1), it was concluded that tetany involves both hypomagnesemia and hypocalcemia. These conclusions are confirmed by the 1957 observations except for the presence of low normal[3] values for serum calcium in the last 4 cases listed. From experience with parturient paresis, the assumption was made that the degree of hypocalcemia would be an index of the ability to stand. When an attempt was made to correlate increased motor irritation and other nervous signs with decreased serum magnesium values, some of the case histories agreed with these patterns, but others indicated a more complex relationship. Study of tables 1 and 2 indicates that the calcium to magnesium ratio may be a better explanation of clinical behavior. Quiet or paretic cows usually have low calcium to magnesium ratios. They tend to approach the 4.7 to 1.0 ratio between the low normal value for serum calcium, 8.5 mg./100 ml., and the low normal value for serum magnesium, 1.8 mg./100 ml.[3]

It should be recalled that calcium is known to be related to the irritability of muscle and nerve[6] and that, in the absence of an adequate serum calcium level, the nervous system fails and paresis results. It is known that excesses of magnesium depress the central nervous system and have a peripheral curare-like effect,[6] an effect utilized in some intravenous anesthetics.

If hypocalcemia should exist in the presence of normal serum magnesium levels, a relative excess of magnesium would obviously tend to cause narcosis and paresis such as is seen in typical parturient paresis. Tables 1 and 2 show the reverse condition, that of a relative hypercalcemia. On this basis, it is logical to conclude that the nervous signs reported in the case histories are the result of the irritant effects of a relative excess of calcium, as is seen when the calcium to magnesium ratio is large. Tetany signs, however, also result from both relative and absolute hyperpotassemia,[7] a condition seen in cases of wheat poisoning.[9]

The similarity between "grass staggers," "lactation tetany," and wheat poisoning has been discussed.[9] An excess of either potassium or magnesium in the blood stream will deplete the other, and calcium excretion is promoted by increased potassium.[11] Although it has been stated that excess potassium intake does not lead to hypomagnesemia,[5] these relationships strongly indicate the need for evaluation of the role of potassium in the condition described.

SUMMARY

1) Signs of tetany were observed in beef cattle on winter rations.

2) Occurrence of the condition terminated when pasture became available.

3) Concurrent hypocalcemia and hypomagnesemia was indicated by serum analyses.

4) In the absence of data on serum potassium levels, the nervous signs observed were interpreted as being the result of excess serum calcium relative to serum magnesium levels.

5) Treatment and prognosis are discussed.

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When 8 calves were starved from birth for five to ten days, the 4 which were injected with maternal serum withstood starvation and were raised without difficulty. Of the 4 that were given no serum, 2 died.—*Vet. Bull. (Feb., 1958): Item 520.*

Magnesium Deficiency and Tetany

Hypomagnesemic tetany in dairy cows, as it occurs under ordinary farm conditions, is generally not caused by a lack of magnesium in the diet. It is a conditioned magnesium deficiency brought about by substances in the grass which render magnesium less absorbable or otherwise interfere with magnesium metabolism.

Experiments, mainly with sheep, indicate that various inorganic substances, such as sulfur and phosphorus, contain compounds the ingestion of which is followed by a transient but marked fall in the serum magnesium.

Increasing the dietary magnesium is an effective method of combating hypomagnesemic tetany.—*F. Ender et al. in Nord. Vet.-med., 9, (Dec., 1957): 916.*

Availability of Phosphorus for Swine

Drylot feeding experiments with phosphate materials fed to weanling pigs, at Purdue University, showed the phosphorus availability to be about equal in dicalcium phosphate, monocalcium phosphate, and phosphoric acid, followed by steamed bone meal and defluorinated phosphate, the poorest source being soft phosphate in colloidal clay. By feeding the latter, the fluorine content of the femurs was increased 16 to 30 times, and the ash content was reduced.

Feeding a high level of soft phosphate resulted in pitting and decay of the molar teeth.

In one experiment, using a practical corn-soybean oil meal ration, pigs fed steamed bone meal developed markedly more leg weaknesses than the dicalcium phosphate-fed controls but gains were similar. Adding 0.15 per cent in phosphorus (either in dicalcium phosphate or phosphoric acid) to a corn-soybean oil meal ration resulted in a highly significant increase in weight gain and in feed efficiency.—*J. Anim. Sci. (Feb., 1958): 73.*

Phosphorus in Alfalfa Hay.—Feeding trials with sheep and steers showed that alfalfa hay produced on virgin sandy soil and containing only 0.1 per cent phosphorus was unpalatable and resulted in poor gains. The value of the hay was improved by the addition of disodium phosphate. Hay containing 0.15 per cent phosphorus was much more palatable and was equal in feeding value to hay containing greater amounts.—*J. Anim. Sci. (Feb., 1958): 113.*

Parenteral Use of Iron for Anemia in Man.—While the oral administration of iron is effective and is the preferred treatment in man, an iron-dextran complex for intramuscular injection is indicated in the following conditions: (1) where the oral use of iron is contraindicated because of enteritis or active chronic ulcerative colitis; (2) where the maximal rate of

hemoglobin regeneration is needed, such as late in pregnancy; (3) in *infants* having *iron deficiency anemia*; (4) in case of massive hemorrhage where transfusions can not be given. (It is also indicated in other conditions of less consequence in animals.)

Intramuscular administration of iron is poorly effective in immobile patients and those with chronic infections.—*Proc. Mayo Clinic (Dec. 11, 1957): 705.*

Reduced Ration Benefits Swine Production.—A ten-year study with ten generations of swine, a total of 1,705 animals, at Washington State College, revealed that those fed only 70 per cent as much as their full-fed kin were leaner, taller, produced more meat per pound of feed, and weaned more pigs than the fat hogs. However, the latter gained weight faster.—*Successful Farm. (March, 1958): 10.*

Saponins and Bloat in Ruminants

Saponins probably have more than one mode of action. The polysaccharide slime produced by ruminal microorganisms may contribute to formation of a stable froth. There is evidence that alfalfa saponins are utilized by certain rumen bacteria with production of acids, gas, and large amounts of slime. Thus, frothy bloat may be caused by an increase in production of bacterial polysaccharide slime.

Bacterial action on soluble sugars in the plant material probably enhances the quantity as well as the character of the slime in the rumen. This may account for the control of bloat by the use of antibiotics. —*Science (Feb. 14, 1958): 335.*

Parturient Paresis and Vitamin D

An estimated 45,000 to 50,000 cases of parturient paresis occur in Sweden each year. To test its prophylactic effect, a single dose (6 million I.U.) of vitamins D_2 and D_3 plus dihydrotachysterol were given during the week before parturition to cows which had been affected one or several times.

In the first trial, 8 of 28 cows treated intramuscularly (28.6%) developed parturient paresis, compared with 4 of 23 (17.4%) of the controls. When treated intravenously, 8 of 31 cows (25.8%) and 10 of the 26 control cows (38.5%) were affected.

The difference was not considered statistically significant. However, of the 31 cows treated intravenously, 14 (45.1%) retained their fetal membranes compared with only 1 (3.8%) of the controls. The increase was attributed to an abortifacient effect when the drug was given intravenously.—*G. Jonsson in Nord. Vet.-med. (Jan., 1958): 21.*

Copper Nutrition in Ruminants

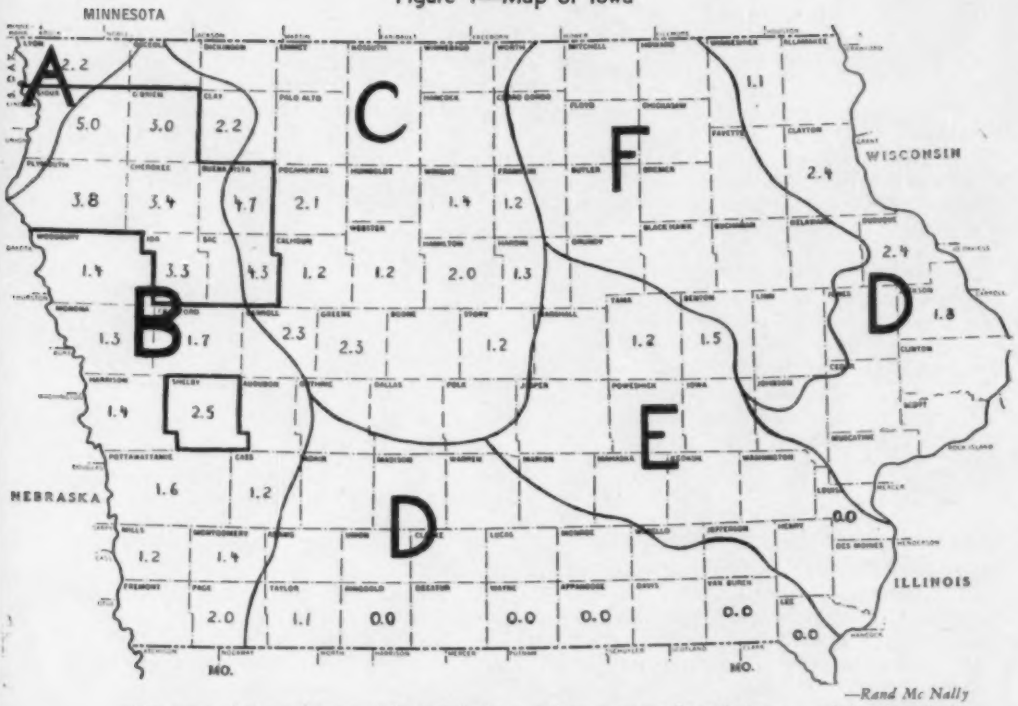
Copper deficiency of cattle and sheep in Australia and New Zealand occurs where the herbage is deficient in copper (4 p.p.m.), but in western Europe, it occurs where the copper content is high by comparison (24 p.p.m.). This indicates that other factors influence the absorption or storage of copper by the animal. The chief signs of copper deficiency are loss of condition and a discolored coat, especially in young cattle, and "swayback" or neonatal ataxia in lambs. Anemia is not characteristic of the condition in Europe as it is in Australia.

Molybdenum and copper are somewhat antagonistic; a moderate excess of molybdenum results in scouring in cattle only when the copper level is low. This antagonism does not explain the deficiency in animals in Britain since there is only 7 p.p.m. of molybdenum, whereas in New Zealand 20 p.p.m. must be present to result in disorders even when there is only 10 p.p.m. of copper.

A third factor is inorganic sulfate which was found to be synergistic with molybdenum in reducing copper stored in a sheep's liver—also in reducing the quality of their wool. However, in Britain, the inorganic sulfate, as well as the copper and molybdenum contents of the herbage, is similar in areas where deficiency does and does not occur. The copper in British pastures may be present in the form of complexes which are not absorbed or, if absorbed, can only be excreted.

In experiments, most of the animals' response was to the copper fraction which was extractable with water, largely in a complex form. In rats, 3 μg. of residual herbage copper per day was equivalent to 10 or 15 μg. of inorganic copper. Some of the inorganic copper may combine with other food components and not be absorbed. This possibility requires more research.—*Nutr. Rev. (March, 1958): 78.*

Figure 1—Map of Iowa



—*Rand Mc Nally*

This map of Iowa (prepared in 1950) shows soil type areas (boundaries=curved lines) and the counties to which the greatest quantities of virulent erysipelas culture was released for use in vaccinating swine in the first ten months of 1950.

The map shows the marked correlation between the alkalinity of the soil and the prevalence of swine erysipelas. The number of tons of lime per acre required by each soil region, in order to grow legumes readily, is as follows: region A (northwest corner)—none; B—0 to 2 tons; C—0 to 3 tons; D—2 to 4 tons; E—3 to 4 tons; and F—3 to 6 tons per acre.

The figure in each county shows the percentage of all culture used in the state which was assigned to veterinarians in that county.* Where no figure is shown, that county used only a fraction of 1 per cent. No culture was used in the seven counties showing a zero. Also shown is the marked lack of correlation between the density of the swine population and the prevalence of erysipelas. The concentration of swine has always been as great in southeastern as in northwestern Iowa, yet one county (Sioux) bordering on South Dakota used as much vaccine as did 30 counties in the southeastern section. The seven counties using the most culture are contiguous (see border line around the group) and lie mostly in the north half of area B; the eighth ranking county (Shelby, 2.5%) is also in area B.

For comparison, the percentage of virulent culture released to each county during 1957 has also been computed.** Even though the practitioners now have a choice of more prophylactic agents, i.e., bacterin and avirulent live culture (see p. 365), the similarity between the figures in 1950 and 1957 is remarkable.

The average percentage used in each county in each soil area is as follows (1957 data in parentheses): area A (parts of 3 counties)—3.67 (3.33); area B—2.26 (2.04); area C—1.08 (0.90); south area D—0.23 (0.28), east area D—0.95 (0.60); area E—0.45 (1.07); and area F—0.47 (0.72). These data indicate a slight increase in 1957 for eastern areas E and F.

*The percentage figures are merely indicative of the prevalence of erysipelas in swine since practitioners used various methods (vaccination with anti-swine erysipelas serum and culture, serum alone, or with anti-hog cholera serum) to control the disease. Also the amount of culture used varied widely with the practitioners' views. Some urged their clients to vaccinate all baby pigs while a few (especially the author who had "burned his fingers" with that method, in Plymouth County in 1944) advised withholding vaccination until the pigs were weaned unless the disease appeared before then.

**We are indebted to Iowa's state veterinarians, Dr. H. U. Garrett in 1950, and Dr. A. L. Sundberg in 1957, for these data.

How Ubiquitous Is the Erysipelas Bacillus?

Most of the qualified persons who have pondered the question believe that erysipelas is a noneradicable disease. This belief is based on the frequency of the finding of the organism—*Erysipelothrix rhusiopathiae*—apparently living as a saprophyte on fish, both fresh water and salt water varieties; in tonsillar and other tissues of apparently healthy animals, especially swine; and the supposition that it can live for long periods, if not permanently, in certain soils.

Van Es[3] stated that in Europe, where erysipelas is common, and probably in parts of this country, "its microbic cause is to a high degree ubiquitous." Also, that the organism exists as a "saprophyte inhabiting the soil, or wherever else nitrogenous substances are decomposing. Under favorable conditions (sandy soils, rich in lime and humus), the bacillus is capable of multiplying in the soil." Also, "in Nebraska most outbreaks . . . are soil-borne."

Hagan states[2] that "In some parts of the corn belt (South Dakota, Nebraska, Iowa), the disease has developed into one of the major problems of the industry as it has been for many years in continental Europe."

The quick-killing, septicemic form of swine erysipelas was recognized for the first time on this continent in the southeastern corner of South Dakota, about 1930. In the next decade, it was most publicized in eastern Nebraska. Since 1931, these areas and northwestern Iowa have comprised the nation's leading enzootic area for the acute disease in swine. As shown on the map (fig. 1), 27 per cent of the erysipelas culture used in Iowa in 1950 (22% in 1957) was used in seven northwestern counties (7%) of the state.

Legumes, which require a relatively alkaline soil, grow most readily in the western extremity of the "swine belt," where erysipelas is "most enzootic." However, this is only circumstantial evidence that the organism survives as a saprophyte in that soil. The Van Es statements, regarding multiplication of the organism in the soil, were based on investigations conducted in Europe. In the first such study we know of on this continent (p. 357), *Ery. rhusiopathiae* did not live long in samples of soil taken from pens where the disease occurred naturally, in Ontario, regardless of the alkalinity of the soil.

While this finding does not mean the organism is not capable of surviving in other soils, it does indicate the need for similar tests in other areas. (Some authorities doubt that soil type affects the incidence of erysipelas in turkeys.)

Observations on Swine Erysipelas

While practicing in Plymouth County, Iowa, from 1931 through 1951, the following peculiarities of the disease were observed. Acute erysipelas would appear in several herds in one neighborhood, almost as if it were contagious, and would recur in the same area for several years, but not regularly on the same farms. It would then disappear and occur in other communities. However, it apparently was not contagious since, in at least two instances, sows died of acute erysipelas, yet their young nursing pigs remained healthy.

As also reported from Germany,[4] the incidence in the area moved in four- to five-year cycles.[1] There were 40 herds with acute erysipelas in 1940, only 11 in 1942, 44 in 1944, only 9 in 1947, then 68 in 1949. Also, vaccination failed to protect chiefly in the "peak" years. This indicated either a cyclic shift in the virulence of the organism, the cyclic presence of some stress factor, or a cyclic variation in acquired resistance from natural exposure.

Many observers believe that stress factors play an important role in the incidence of acute erysipelas. The disease often appears in extremely hot weather (it has been misdiagnosed as "overheating," *see* p. 364), or a day or two after swine have feasted on a carcass or on tankage, or after the first feeding of new corn. The latter may account for the often observed increased incidence of the disease in October.

While these observations neither definitely support nor refute the ubiquity of the organism, or its ability to live long in the soil, they do indicate that it must be present in the animals in order for them to react to stress so suddenly.

References

[1]Aitken, W. A.: Swine Erysipelas. Iowa Vet. (May, 1950): 16.

[2]Hagan, W. A., and Brunner, D. W.: Infectious Diseases of Domestic Animals. 3rd ed. Comstock Publishing Assoc., Ithaca, N. Y. (1957): 165.

[3]Van Es, L.: Swine Erysipelas Infection in Man. Univ. of Nebraska Bull., *130*, (1942): 3.

[4]Van Es, L., and McGrath, C. B.: Swine Erysipelas. Univ. of Nebraska Bull., *84*, (1936): 10.

Current Literature

ABSTRACTS

Histomoniasis in Turkeys

Typical gross lesions were observed in the ceca, livers, spleens, kidneys, and lungs of turkeys experimentally infected with *Histomonas meleagridis*.

Microscopic lesions in which the parasite could be demonstrated were observed in the ceca, livers, kidneys, and spleens. The lesions were characterized by hyperemia, hemorrhage, lymphocytic infiltration, macrophages, the presence of multinucleated giant cells, necrosis and, usually, a serous exudate. This was a granulomatous type of inflammation, accompanied by necrosis.

In the lungs, pancreas, and heart, there were areas of hyperemia and serocellular exudate, but parasites were not demonstrated. Lymphocytic infiltration was observed in the lungs.

The control birds remained healthy, and no gross or microscopic lesions were found at necropsy.—[*Thomas D. Malewitz, Russell A. Runnells, and M. Lois Calhoun: The Pathology of Experimentally Produced Histomoniasis in Turkeys. Am. J. Vet. Res., 19, (Jan., 1958): 181-185.*]

Blood Pressure of the Turkey

Measurements made by cannulation of the carotid arteries of unanesthetized Jersey Buff poults showed that there were no sex differences in the blood pressures of 6- to 7-week-old poults. Averages were: systolic pressure, 193 ± 3.6; diastolic pressure, 150 ± 2.8; pulse pressure, 43 ± 1.9; and heart rate, 306 ± 4.8.

Systolic and pulse pressures of 8- to 9-month-old adult turkeys were greater and heart rates less than in poults. The pulse pressure of the adult male (75) was significantly higher than that of the female (55), whereas the heart rate of the female (185) was significantly higher than that of the male (144).

Turkey pressures, in general, were higher than those reported previously for the chicken, duck, and pigeon. The adult male pressure pattern appeared to reflect decreased distensibility, such as might be expected from aortic atheromatosis; the elevated pressures, in turn, may initiate spontaneous aneurysms and ruptures of the atherosclerotic vessels, particularly in the male.—[*Harold S. Weiss and Mary Sheahan: The Influence of Maturity and Sex on the Blood Pressure of the Turkey. Am. J. Vet. Res., 19, (Jan., 1958): 209-211.*]

Growth of Newcastle Disease Virus

Growth of Newcastle disease virus in tissue culture was affected by changes in hydrogen ion concentration and in the concentration of potassium, calcium, and magnesium ions.

The maximal titer of Newcastle disease virus (Roakin strain) was obtained in cultures of chorioallantoic membranes suspended in Tyrode's solution at pH 9.5; the smallest yield was obtained at pH 2.7. High yields of virus were obtained when the pH range of Tyrode's solution was 6.7 to 7.6.

The maximal titer of Newcastle disease virus was obtained from growth in chorioallantoic membranes incubated in Tyrode's solution that contained 0.2 Gm. of $CaCl_2$ per liter. Greater concentrations of $CaCl_2$ appeared to deter growth of virus in tissue culture.

Potassium was found to be essential for growth of this virus in tissue cultures. Maximum yields of virus were obtained in membranes suspended in Tyrode's solution that contained 0.4 Gm. of KCl per liter. Virus growth was inhibited in membranes incubated in Tyrode's solutions that were deficient in potassium. This inhibiting effect could be overcome by addition of potassium to the solution. Magnesium ions did not overcome the inhibition of growth of virus in tissues suspended in Tyrode's solution deficient in potassium and were detrimental to the production of virus in tissue culture when the magnesium exceeded the concentration normally found in Tyrode's solution.

Increasing the concentration of glucose in Tyrode's solution produced an inhibitory effect upon the growth of virus in tissues suspended in these solutions.—[*F. Zuschek, R. P. Hanson, and C. A. Brandly: Factors Influencing the Growth of Newcastle Disease Virus in Isolated Chorioallantoic Membranes. Am. J. Vet. Res., 19, (Jan., 1958): 191-195.*]

Lesions of Newcastle Disease

Newcastle disease was induced in susceptible, 5-week-old chickens by the intramuscular injection of California strain 11914 Newcastle disease virus. All inoculated chickens died of the disease or were killed within 96 hours. The signs of Newcastle disease in these birds were drowsiness, tremors, inability to maintain the posture of the head and, finally, paralysis and coma. The birds were unable to maintain their normal attitudes after the appearance of the tremors.

The predominant tissue change was an extensive hyperemia of the central nervous system, accompanied by proliferation of the endothelial cells. Degenerative changes of the neurons, ranging from mild chromatolysis to frank necrosis, were found in all cases, primarily in the vestibular, reticular, and cerebellar nuclei, and in the Purkinje cells.—[*D. J. Sullivan: Lesions in the Cerebellum and in the Reticular and Vestibular Centers in Newcastle Disease. Am. J. Vet. Res., 19, (Jan., 1958): 186-190.*]

Cytopathology of Infectious Canine Hepatitis

Cytological changes are described as they occur in canine kidney cell cultures inoculated with infectious canine hepatitis virus.

The first definite change was the appearance of intranuclear inclusion bodies, beginning at about 23 hours after cells were exposed to virus. The number of cells containing inclusions increased

after this time. As the infection progressed, cells died and large gaps appeared in the cell sheet.

Estimates of the rate of virus propagation were made by assay of fluids surrounding infected cells at intervals after cells were inoculated.—[*R. W. Leader: Cytopathology and Rate of Release of Infectious Canine Hepatitis Virus Grown in Dog Kidney Cells. Am. J. Vet. Res., 19, (Jan., 1958): 152-158.*]

Canine Mastocytoma

The literature revealed reports of 343 mast cell tumors of the skin of dogs and eight in other species, including man. In the collection studied, mastocytomas comprised 20 per cent of the primary cutaneous canine tumors. The mastocytomas invaded locally, metastasized, and recurred following surgical excision, thus justifying the term "neoplasm" for this process. The mastocytomas had a tendency to become eroded, grew without demarcation, and had a site preference for the external genitalia (15%) and hindquarters (50%).

The Boston Terrier exhibited a marked, and the Boxer a lesser, breed predisposition for this tumor. Males and females were affected with equal frequency. Most mastocytomas occurred in dogs over 6 years of age. Of the tumors, 28 per cent were malignant, 17 per cent recurred between two weeks and one year, and 16 per cent metastasized to inguinal nodes, spleen, liver, and sublumbar nodes. Multiple occurrence was present in 21 per cent. In no instance did internal mastocytomas occur in the absence of cutaneous tumors.

Microscopically, the mastocytoma was characterized by a diffuse proliferation of freely situated round cells of uniform size and shape. With proper stains, a varying number of metachromatic granules were seen in their cytoplasm. Generally, rapidly growing mastocytomas, consisting of immature mast cells, contained only fine dustlike granules, while mature cells had an abundance of coarse granules.—[*S. W. Nielsen and C. R. Cole: Canine Mastocytoma—A Report of One Hundred Cases. Am. J. Vet. Res., 19, (Jan., 1958): 417-432.*]

Serum Proteins of the Guinea Pig

Guinea pig serum, when heated at 56 C. for 30 minutes, and separated by paper electrophoresis in barbital buffer (pH 8.6, 0.05 ionic strength), shows decreases in the concentrations of albumin, *beta* and *gamma* globulins, and an increase in *alpha* globulin. When hemoglobin is added to fresh serum, the concentration of albumin decreases, whereas increases in the *alpha* and *beta* globulins occur; the *gamma* globulin is unaffected. This phenomenon is accentuated when serum-hemoglobin mixtures are heated.

Guinea pig hemoglobin appears to be a single entity having the mobility of *alpha* globulin. The increases in concentrations of *alpha* and *beta* globulins of the serum-hemoglobin mixtures do not coincide with the homogeneous electrophoretic mobility of hemoglobin alone and it appears that complexes are formed.—[*G. T. Dimopoullos and O. N. Fellowes: The Effects of Heat and Hemoglobin on the Serum Proteins of the Guinea Pig. Am. J. Vet. Res., 19, (Jan., 1958): 230-232.*]

Cerebrospinal Fluid Pressure

The cerebrospinal fluid pressures of dwarf beef cattle ranged from 268 to 700 mm. when measured at the atlanto-occipital articulation; from 140 to 560 mm. when measured at the juncture of the last lumbar vertebra and the sacrum. In a group of normal beef and dairy cattle presumed to be free of dwarfism, no positive pressures could be obtained at the lumbosacral juncture. Of 53 beef calves in a herd in which dwarf carriers could have been present, 29 cattle exhibited positive pressures in the lumbar region. It does not appear, however, that animals carrying the dwarf gene can be detected by the measurement of cerebrospinal fluid pressure.—[*E. J. M. Fransen and F. N. Andrews: Cerebrospinal Fluid Pressures in Dwarf and Normal Cattle. Am. J. Vet. Res., 19, (Jan., 1958): 336-337.*]

Cholesterol Levels in Normal and Dwarf Cattle

The blood plasma cholesterol levels of normal nonpregnant female and castrated male beef cattle averaged 102.5 ± 2.0 mg./100 ml., in comparison with 81.1 ± 3.6 for male and female dwarf beef cattle. The difference was significant. The cholesterol levels were higher in dwarf bulls than in dwarf females. There were no significant seasonal differences; however, following a severe cold spell there was some reduction in blood cholesterol levels of both normal and dwarf cattle.—[*J. M. Fransen and F. N. Andrews: Blood Plasma Cholesterol Levels in Normal and Dwarf Beef Cattle. Am. J. Vet. Res., 19, (Jan., 1958): 332-335.*]

Influence of Season on Cholesterol in Ewes

Over a two-year period, observations were made on the blood cholesterol levels of sexually mature ewes under a normal fluctuating environment and under partial environmental control. There were no significant seasonal differences in ewes in a normal environment. The alteration of day length, or artificial cooling during the summer months, had no influence on blood cholesterol levels, and there were no significant differences in pregnant and nonpregnant ewes.—[*T. M. Means and F. N. Andrews: The Influence of Season on Blood Plasma Cholesterol in the Ewe. Am. J. Vet. Res., 19, (Jan., 1958): 295-298.*]

Sulfaquinoxaline and Rabbit Mortality

The effects of feeding 0.025 per cent sulfaquinoxaline were studied on 20 white New Zealand does and 367 young. The drug had no effect on total enteritis mortality, but it did reduce the hemorrhagic type of enteritis by 70 per cent. Deaths due to primary pneumonia were elimi-

nated, and the number of *Pasteurella multocida* isolations were reduced by 70 per cent. Nest-box mortality remained unchanged. No toxic effects were noticed during the test, and the sulfaquinoxaline ration was palatable.—[*Karl W. Hagen: The Effects of Continuous Sulfaquinoxaline Feeding on Rabbit Mortality. Am. J. Vet. Res., 19, (Jan., 1958): 494-496.*]

BOOKS AND REPORTS

Veterinary Anesthesia

The fourth edition of this book covers the use of anesthetics, local and general, in most of the domestic animals encountered in practice. New advances in the field of human anesthesia, such as have occurred in the last few years, generally have an effect in the field of veterinary anesthesia. The author has included in this book information on new sedative and analgesic drugs as well as new ultra-short acting barbiturates. Chapters on inhalation anesthetics offer additional information on the closed-type of inhalation anesthesia in the dog.

The chapter on curare and drugs with curare-like action is certainly valuable reference information for any veterinarian attempting this type of anesthesia.

The veterinarian performing either minor or major surgery could well afford to add this informative book on anesthesia to his or her medical library.—[*Veterinary Anaesthesia. By John G. Wright. 4th ed. 317 pages. Williams & Wilkins Co., Mt. Royal Ave., Baltimore 2, Md. 1957. Price $6.75.*]—P. J. MEGINNIS.

Handbook of Histology

This handbook was prepared to simplify the recognition of sections of tissues and organs by students of microscopic anatomy. It is organized in outline form and contains several summary charts and uncomplicated line diagrams.

The first three editions were widely adopted in histology courses given in zoology departments and medical schools. The fourth edition has been thoroughly revised and appreciably expanded. New material on the reticuloendothelial system, the interpretation of sections by visualizing in the third dimension, and several other subjects have been introduced. The glossary has been expanded and made self-pronouncing.

Although most of the information is applicable to mammals in general, there are a few areas which apply specifically to primates. Specializations found in domestic animals are not included.

Only the salient features of tissues and organs are presented. Controversial theories are passed over lightly or omitted entirely, presumably but not necessarily to the advantage of the busy student. Features essential to differential diagnosis are emphasized in italics.

The handbook should be helpful to students provided it is used for the purpose intended by the author: that of supplementing rather than supplanting a classical histology text. In the reviewer's experience, some students in an effort to avoid unnecessary reading have used this handbook to their ultimate disadvantage, mistaking a mastery of thumb rules for true understanding. This possible disadvantage, however, is not the fault of the handbook, which does not purport to be a complete text. On the whole, it is a well prepared, worthwhile aid.—[*Handbook of Histology. By Karl A. Stiles. 4th ed. 240 pages; 10 summary tables; 8 pages of line diagrams. Blakiston Co., 1021 Walnut St., Philadelphia 5, Pa. 1956. Price $3.00.*]—A. H. SAFANIE.

Microtechniques of Clinical Chemistry

Research workers concerned with the effect of diseases or drugs on the human organism or on small laboratory animals will find this book of value. It contains chapters on principles commonly used in microchemistry, units of measurement used in microanalysis, measuring and weighing samples, and an extensive section on methodology. The appendix contains information on density, surface tension, normal values, evaluating test data, absorption spectrophotometry, and chelate compounds.—[*Microtechniques of Clinical Chemistry for the Routine Laboratory. By Samuel Natelson. 484 pages; illustrated. Charles C Thomas, 301-327 E. Lawrence Ave., Springfield, Ill, 1957. Price $11.00.*]

Uses of Epidemiology

Written primarily for students of clinical and preventive medicine and for laboratory workers and clinicians, this book discusses epidemiology as it relates to public health, including the work of the health services, identification of syndromes, and the search for causes of epidemics.—[*Uses of Epidemiology. By J. N. Morris. 135 pages. Williams and Wilkins Co., Mount Royal and Guilford Aves., Baltimore 2, Md. 1957. Price $4.00.*]

Literature of Agricultural Research

This bibliography includes sections on agriculture in general, plant sciences, animal sciences (including a section on veterinary medicine), physical sciences, food and nutrition, and social sciences as they pertain to agriculture.—[*Literature of Agricultural Research. By J. Richard Blanchard and Harald Ostvold. 231 pages. University of California Press, Berkeley 4, Calif. 1958. Price $5.00.*]

The Springer Spaniel for Show and Field

In addition to chapters on general care, feeding, house training, and history of the Springer Spaniel, this book gives the new official standards for this breed and discusses training for both the show ring and hunting.—[*The Springer Spaniel for Show and Field. By Maxwell Riddle. 80 pages; 155 illustrations. Judy Publishing Co., 3323 Michigan Blvd., Chicago 16, Ill. 1957. Price $4.00.*]

THE NEWS

Third World Congress on Fertility and Sterility Scheduled for 1959

The Third World Congress of Fertility and Sterility, sponsored by the International Fertility Association, will be held in Amsterdam, Holland, June 7-13, 1959.

The section programs will include reports on various aspects of male and female sterility, basic research, animal reproduction, and psychosexual problems. Priority will be given to original reports on embryonic death, hormonal factor, techniques for study of endocrine functions, biochemistry of spermatogenesis, and psychosexual problems in sterility.

Titles for papers should be sent not later than June, 1958, to both Dr. Alfonso Alvarez-Bravo, Avenida Horacio 1506, Mexico City 5, D. F., and Prof. Dr. B. S. ten Berge, Academisch Ziekenhuis, Holland. Official languages, with simultaneous translation, will be English, French, German, and Spanish.

For further information and registration, apply to Dr. L. I. Swaab, Sint Agnietenstraat 4, Amsterdam, Holland.

Examination — American College of Veterinary Pathologists

Applications now may be submitted for the 1958 examination of the American College of Veterinary Pathologists. These applications must be in the hands of the secretary-treasurer by July 20, 1958, in order to be considered by the council of this organization which meets during the AVMA meeting in Philadelphia. It is expected that the examination will be given on Nov. 28, 1958, in Chicago.

To meet the prerequisites for this examination, a candidate must:

a) Have satisfactory moral and ethical standing in the profession;

b) Be a graduate of a veterinary school recognized by the American Veterinary Medical Association;

c) Be a member in good standing of the American Veterinary Medical Association;

d) Have qualified at some time to practice veterinary medicine either by successfully completing the national or any state board examination;

e) Have completed at least five calendar years of professional experience, exclusive of reasonable vacation periods, subsequent to graduation from veterinary school, at least two years of which shall be supervised training in pathology acceptable to the council. At least one of the additional three years of professional experience shall be in teaching, research or practice of veterinary pathology;

f) Submit to the council a detailed statement of his qualifications, including references of any publications, and other evidences of his professional experience and competence.

Further details and application forms may be secured by writing to: T. C. Jones, D.V.M., Secretary-Treasurer, 180 Longwood Ave., Boston 15, Mass.

Current Officers of Association of State Public Health Veterinarians

The new officers of the Association of State Public Health Veterinarians are: Dr. Ernest J. Witte, Pennsylvania Department of Health, Harrisburg, president; Dr. E. R. Price, Missouri Division of Health, Jefferson City, vice president; Dr. Oscar Sussman, New Jersey State Department of Health, Trenton, secretary-treasurer; Dr. R. L. Hectorne, Kentucky Department of Health, Louisville, and Dr. S. L. Hendricks, Iowa State Department of Health, Des Moines, executive committee members.

s/Oscar Sussman, D.V.M., *Secretary-Treasurer.*

Course in Epidemiology and Control of Food-Borne Diseases

A multi-discipline course in epidemiology and control of food-borne diseases will be offered at the Communicable Disease Center, Public Health Service, Atlanta, Ga., May 19-23, 1958.

Designed to provide public health workers with tools for preventing and controlling food-borne diseases, the course is offered for physicians, veterinarians, nutritionists, laboratory workers, and other members of the public health team.

Preference will be given to persons whose professional tasks involve the application of epidemiological techniques in this field, and registrants will be expected to attend all sessions.

Further information and application forms may be obtained from: Chief, Communicable Disease Center, Public Health Service, 50 Seventh St., N.E., Atlanta 23, Ga. Attention: Chief Training Branch.

AMONG THE STATES AND PROVINCES

Alabama

Alabama Annual Meeting.—The fifty-first annual meeting of the Alabama Veterinary Medical Association was held March 16-18, 1958, at the Stafford Hotel in Tuscaloosa. Drs. R. O. Moore, Jr., and J. T. Phifer were chair-

man and co-chairmen of the local committee.

A panel on large animal medicine was moderated by Dr. F. G. Schell, professor, large animal surgery, Alabama Polytechnic Institute, with Drs. H. F. Findley, L. E. Irby, and J. L. Wesley, all Alabama practitioners, participating.

Other subjects dealing with the treatment of large animals were presented by Dr. W. J. Gibbons, professor, medicine and infectious diseases at the Institute, who discussed cattle diseases; Dr. Jacques Jenny, head, large and small animal clinics, University of Pennsylvania, spoke on orthopedics of large animals; and Dr. C. D. Van Houweling, Washington, D. C., outlined the veterinarian's responsibility to the livestock producer.

The brucellosis program in Alabama was discussed by Dr. R. N. Lauderdale, ARS, Montgomery.

Dr. G. V. Kenmore, practitioner of Montgomery, moderated a panel on small animal medicine, with Drs. J. L. Chambers, C. L. Holloway, W. W. Kent, W. P. Monroe, C. B. Phillips, and R. A. Kirby, all Alabama practitioners, participating.

Dr. C. E. DeCamp, Pitman-Moore Company, Indianapolis, spoke on "Cats in Your Practice," and "Care of the Older Dog."

Hospital management and surgical techniques were discussed by Dr. G. M. Young, small animal practitioner of Jacksonville, Fla., and Mr. W. C. Sugg, of the Upjohn Co., Kalamazoo, Mich., talked about the relationship of the pharmacist and the veterinarian.

Dr. H. E. Kingman, Jr., assistant executive secretary of the AVMA, told how the public relations activities of the AVMA and the local associations are coordinated.

On the evening of March 17, the members and their wives attended a smorgasbord dinner, followed by entertainment and dancing.

California

Southern California V.M.A. Meeting.—The regular meeting of the Southern California V.M.A. was held on March 19, 1958, at the Rodger Young Auditorium in Los Angeles.

Mr. Everett A. Hart, attorney and trust officer of the Title Insurance and Trust Co., Los Angeles, spoke on "Tax Savings Through Estate Planning." Many requests had been received by the program committee for this type of talk, and the committee felt fortunate to obtain Mr. Hart for this dinner meeting.

Colorado

Nineteenth Annual Conference.—The nineteenth annual conference for veterinarians was held at the College of Veterinary Medicine, Colorado State University, Fort Collins, on Feb. 17-19, 1958.

Out-of-state speakers and their subjects were: Drs. M. G. Fincher—Mastitis Control; Dairy Cattle Disease Problems; George W. Mather—Canine Leptospirosis; Poisoning in Small Animals; Hadleigh L. Marsh, Feedlot Diseases of Cattle; Swine Disease; Blaine McGowan—Polyarthritis in Lambs; Epididymitis of Rams; W. V. Dakin—Progress and Efficiency of Thoroughbred Breeding.

A clinicopathological panel on case reports from the university small animal clinic was moderated by Dr. W. B. Lumb; participating in the panel were Drs. Maxine Benjamin, Homer Brown, Lee Phillips, Francis T. Candlin.

Dr. Ben Shambaugh, state veterinarian, discussed livestock disease control in Colorado.

Among the speakers from the College of Veterinary Medicine at Colorado State University were: Drs. W. D. Carlson, H. J. Hill, Rue Jensen, Jean Flint, A. F. Alexander, R. H. Udall, John R. Collier. Their subjects covered laboratory diagnostic techniques, interpretation of radiographs of large and small animals, large and small animal electrocardiograms and phonocardiograms, and various diseases in swine, cattle, and sheep.

Surgical demonstrations were presented as follows: Celiotomy in the Horse by Dr. W. Aanes; Cesarean or Urethral Calculi Correction by Dr. James T. Ingram; Rumenotomy by Dr. S. W. Dawley; and Thoracic Surgery by Dr. W. V. Lumb.

At the banquet and dance on February 18, Dr. Vyrle D. Stauffer, of Arvada, was the speaker, substituting for Dr. R. E. Rebrassier, AVMA president-elect, who was ill.

Dean Rue Jensen presented a plaque from the Colorado V.M.A. to Mr. G. Norman Winder of Craig and Denver, Colo., in recognition of his contributions to the veterinary profession.

District of Columbia

Second Quarterly Meeting.—The District of Columbia V.M.A. held its second quarterly meeting in mid-March in the Dart Auditorium of the Armed Forces Institute of Pathology, Walter Reed Army Medical Center. About 90 members and their guests were present.

Following a short business meeting, Dr. W. W. Armistead, president of the AVMA, delivered an address on the future of the veterinary profession.

s/WILLIAM I. GAY, *Secretary-Treasurer.*

Illinois

Eastern Illinois Meeting.—The regularly scheduled meeting of the Eastern Illinois Veterinary Medical Association was held at the Hotel Tilden Hall, Champaign, on March 6, 1958.

Dr. D. R. Lingard, College of Veterinary Medicine, University of Illinois, spoke on pleuropneumonia in swine and Dr. A. B.

Hoerlein, also of the University of Illinois, discussed virus pneumonia in pigs.

s/A. G. SCHILLER, *Secretary-Treasurer.*

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Chicago V.M.A. Meeting.—The March meeting of the Chicago V.M.A. was held on March 11, 1958, at the LaSalle Hotel.

Dr. Robert Knowles, of Miami, Fla., well-known veterinary practitioner, presented an illustrated talk entitled "Professional Parfait, or Tricks of the Trade."

Kansas

Symposium on Rabies.—An interprofessional symposium on rabies will be held on April 18, 1958, at the School of Medicine, University of Kansas, Kansas City, Kan. Both physicians and veterinarians will participate as speakers.

The veterinarians on the program are Dr. Donald J. Dean, Albany, N.Y.; Robert J. Kissling, Montgomery, Ala.; Charles C. Rife, Atlanta, Ga.; James H. Steele, Atlanta, Ga., and Ernest S. Tierkel, Atlanta, Ga.

• • •

Kansas City V.M.A. Meeting.—The Kansas City Veterinary Medical Association held their regular monthly meeting on March 20, 1958, in the Hotel President, Kansas City. A discussion was conducted on the integration into the profession of the summer veterinary student employee and the new veterinary graduate.

Participating in the discussion were Drs. Tom Engle, Parkville, Mo.; James K. Farrell, Boonville, Mo.; John S. Haley, Topeka, Kan.; Merle L. Hendrikson, Emporia, Kan., William L. Schondelmeyer, Independence, Mo. Dr. Jack Knowles, a practitioner of Miami, Fla., the guest speaker, participated in the discussion.

The deans of the veterinary medical schools of Kansas State College and the University of Missouri, together with veterinary medical upper classmen, were invited to attend.

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Dr. and Mrs. Earl N. Moore, on a Kansas State College Mission to India, ride through the jungles on elephants. Mrs. Moore is shown here atop, according to Mrs. Moore, a "lovely" elephant.

Michigan

1958 Officers of the Women's Auxiliary to the Michiana V.M.A.

Left to right—Mrs. George Freier, Benton Harbor, Mich., vice-president; Mrs. L. G. Ramsey, LaPorte, Ind., president; Mrs. Klaus Friedburg, Three Oaks, Mich., secretary (seated); and Mrs. A. C. Dufour, Elkhart, Ind., treasurer.

Nevada

Nevada State V. M. A. Annual Meeting.—The Nevada State Veterinary Medical Association held its annual meeting at the Holiday Hotel in Reno, Feb. 27 to March 1, 1958.

Dr. R. M. Cello, University of California, and Dr. M. H. Schaffner, Santa Rosa, Calif., presented timely talks on small animal medicine and surgery.

Dr. J. A. Howarth, University of California, discussed methods of increasing reproductive efficiency in commercial beef herds, and presented a paper on leptospirosis.

Dr. J. D. Wheat, University of California, gave an illustrated review of the means of diagnosis of the many lamenesses of the horse, which was enthusiastically received by those in attendance.

Dr. Kenneth L. Kuttler, University of Nevada, reviewed the research activities being conducted in veterinary science at the University of Nevada.

Dr. John L. O'Harra, Nevada Department of Agriculture, detailed experimental findings in studies of injecting copper glycinate subcutaneously into cattle to combat molybdenum poisoning.

Dr. W. F. Fisher, Nevada Department of Agriculture, discussed the disease regulatory program in effect in the State of Nevada.

Dr. E. E. Maas, ARS, reported on the progress of the brucellosis-eradication campaign in the state.

In business session at the close of the meet-

ing, Dr. F. N. Neville, Winnemucca, turned the gavel over to the newly elected president, Dr. M. L. Houston, of Yerington. Other officers elected for the coming year were Drs. F. Robert Etor, Las Vegas, vice-president; John L. O'Harra, Reno, secretary-treasurer; W. F. Fisher, Reno, delegate to the AVMA House of Representatives.

It was voted to hold the 1959 meeting in Las Vegas.

s/JOHN L. O'HARRA, *Secretary-Treasurer.*

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Women's Auxiliary Meeting.—The Women's Auxiliary to the Nevada V.M.A. held its annual meeting on Feb. 28, 1958, at the Mapes Hotel in Reno.

The new officers elected for the coming year were Mrs. M. L. Houston, Yerington, president; Mrs. F. Robert Stor, Las Vegas, vice-president; Mrs. John L. O'Harra, Reno, secretary; and Mrs. W. F. Fisher, Reno, delegate to the house of representatives of the AVMA Women's Auxiliary.

Members of the Auxiliary and their husbands attended a banquet in the evening, which was followed by dancing.

New Jersey

New Officers of New Jersey V.M.A.—The New Jersey Veterinary Medical Association, at their meeting on Feb. 12-13, 1958 (*see* March 15 JOURNAL for report of meeting), elected the following officers: Drs. O. Sussman, Princeton, president; M. S. Arlein, Maplewood, president-elect; R. L. Ticehurst, Red Bank, vice-president; J. R. McCoy, New Brunswick, secretary; A. F. North, Somerville, treasurer.

The members of the executive committee for the current year are Drs. O. K. Fox, Moorestown; C. Moon, Yardville; J. R. Porteus, Trenton; J. W. Rich, Kearny; C. L. Schenholm, Flemington; R. R. Shomer, Teaneck; M. L. Ziskind, Secaucus.

s/J. R. PORTEUS, *Member Executive Committee.*

Nova Scotia

Nova Scotia Veterinary Association.—Members of the Nova Scotia V.A. attended a short course on basic radiation monitoring at the Civil Defense headquarters on Feb. 21, 1958. Attending were: Drs. K. R. Ainslie, Halifax; A. R. Mitton, Dartmouth; R. W. Kennedy, Sydney River; K. W. Trenholm, Kentville; J. O'Neil, Halifax; and L. J. Dolan, A. R. Ross, E. E. I. Hancock, A. R. Main, H. Van Zwol, all of Truro.

New York

Dr. Mohler's Work Honored.—A bronze plaque (see cut) of Dr. John R. Mohler commemorates his contribution to, and his administration of, research and animal disease control in the U.S.D.A. The plaque hangs in the entry foyer of Laboratory 101 of the Plum Island Animal Disease Laboratory on Long Island.

Dr. John R. Mohler (1875-1953)

Ontario

Central Canada V.A. Meeting.—The fifty-fifth annual meeting of the Central Canada Veterinary Association was held March 21-22, at the Hotel Manitonna in Brockville.

Speakers on the large animal section of the program were Dr. T. W. Carvell, who discussed the control and treatment of mastitis from a practitioner's standpoint; Dr. W. A. Moynihan, Canada Department of Agriculture, told of Canada's brucellosis-eradication program; Dr. A. S. Greig, of the Animal Diseases Research Institute, Hull, Que., spoke on the mucosal disease complex in cattle; and large animal surgery was demonstrated by Dr. F. J. Milne, of the Ontario Veterinary College, Guelph.

In the small animal field, "New Conceptions of Canine Distemper Immunization" was the title of a talk by Dr. R. V. Johnston, of the Pitman-Moore Company, Indianapolis, Ind.; Dr. R. M. Armitage, Shawville, Que., spoke on "Rabies as Viewed in the Field;" and Dr. Claude Kealey, of Ottawa, discussed urinary tract infections in small animals.

A banquet, held on the evening of March 22, was attended by members and their wives.

s/C. K. HETHERINGTON, *Secretary.*

Pennsylvania

Bucks-Montgomery V.M.A. Meeting.—A regular meeting of the Bucks-Montgomery Veterinary Medical Association was held

March 12, 1958, in the Moose Lodge at Doylestown.

Dr. John D. Beck, chief of staff of the Speyer Hospital in New York City, spoke on "The Problems in the Management of a Small Animal Hospital."

s/M. J. DEUBLER, *Secretary.*

South Carolina

Twin Carolinas V.M.A. Officers.—The new officers of the Twin Carolinas Veterinary Medical Association are Drs. D. M. Bedell, Bennettsville, S. Car., president; H. W. Armstrong, Monroe, N. Car., vice-president; J. E. Currie, Southern Pines, N. Car., secretary. The directors of the association are Drs. R. E. Gandy, Rockingham, N. Car.; G. J. Lawhorn, Jr., Hartsville, S. Car.; R. G. Knight, Fayetteville, N. Car.

The meeting date of the association has been changed to the third Friday of each month at the Orange Bowl restaurant, Rockingham, N. Car., at 7:30 p.m.

STATE BOARD EXAMINATIONS

Interested persons can obtain information about applications, fees, deadlines for filing applications, and exact time and place of examinations of the respective boards by writing to the persons whose names and addresses are given below.

BRITISH COLUMBIA—June and November, 1958 (usually end of the second week); Vancouver. G. L. Stovell, British Columbia Veterinary Association, 3187 West 43rd Ave., Vancouver 13, secretary.

DISTRICT OF COLUMBIA—June 26, 1958, Department of Occupations and Professions, 1740 Massachusetts Ave., N.W., Washington, D.C. A. F. McEwan, secretary, 1740 Massachusetts Ave., N.W., Washington 6, D.C.

FLORIDA—June 16-17-18, 1958, Biscayne Terrace Hotel, Miami. Dr. E. L. Matthews, secretary, Box 141, Palatka, Fla.

INDIANA—July 8-9, 1958, House of Representatives, State House, Indianapolis, Ind. Joe W. Green, secretary, Room 413, 611 N. Park Ave., Indianapolis 4, Ind.

IOWA—June 16-17, 1958, Office of the Division of Animal Industry, State House, Des Moines, Iowa. A. L. Sundberg, chief, Division of Animal Industry, Des Moines, Iowa.

KANSAS—May 27-29, 1958, Dykstra Veterinary Hospital, Kansas State College, Manhattan, Kan. Applications must be filed by May 1, 1958. Chas. W. Bower, secretary, 1128 Kansas Ave., Topeka, Kan.

KENTUCKY—July 28, 1958. University of Kentucky, Lexington. J. K. Bushnell, secretary, 229 Houston, Paris, Ky.

MAINE—July 7-8, 1958, State House, Augusta, Maine. E. C. Moore, secretary, Turner Center, Maine.

MARYLAND—June 18, 1958, written examination; June 19, 1958, practical examination; College Park, Md. Harold S. Gober, secretary, 5400 Park Heights Ave., Baltimore 15, Md.

MASSACHUSETTS—June 26-27-28, 1958, Amherst. Dr. Edward A. Blake, secretary, Room 33, State House, Boston, Mass.

MICHIGAN—June 9-10, 1958, Lansing, Mich. Written, practical, and oral examinations. Applications must be on file at least 15 days before examination, accompanied by $25 fee. Address: State Veterinarian, 641 Lewis Cass Bldg., Lansing 13, Mich.

MISSISSIPPI—June 17, 1958, Jackson, Miss. Wm. L. Gates, secretary, Box 417, Clarksdale, Miss.

MISSOURI—June 4-5, 1958, Veterinary Clinic, University of Missouri, Columbia, Mo. Dr. L. A. Rosner, chairman, Missouri Veterinary Board, Box 630, Jefferson City, Mo.

MONTANA—June 30-July 2, 1958, Montana State College, Bozeman. J. W. Safford, secretary treasurer, Capitol Station, Helena, Mont.

NEW YORK—June 18, 19, 1958, practical examination, Ithaca; June 24-27, 1958, written examination, New York City, Albany, Syracuse, Buffalo, and Rochester. John W. Paige, chief, Bureau of Examinations and Registrations, 23 S. Pearl St., Albany, N.Y.

NORTH CAROLINA—June 23-25, 1958, Hotel Washington Duke, Durham, N. Car. James I. Cornwell, secretary, 65 Beverly Road, Beverly Hills, Asheville, N. Car.

OHIO—June 10-11, 1958, College of Veterinary Medicine, Ohio State University, Columbus. H. G. Geyer, DVM, secretary, Room 720, Ohio Department Building, 65 S. Front Street, Columbus 15, Ohio.

OKLAHOMA—May 20-22, 1958, College of Veterinary Medicine, Oklahoma State University, Stillwater. Dr. Robert S. Todd, secretary-treasurer, Oklahoma Board of Veterinary Medical Examiners, 310 State Capitol, Oklahoma City, Okla.

ONTARIO—June 9-11, 1958, Ontario Veterinary College, Guelph, Ont., G. A. Edge, secretary, Box 37, Postal Station F, Toronto 5, Ont.

RHODE ISLAND—July 8-9, 1958, 505 Veterans' Memorial Bldg., Providence, R.I. T. J. Grennan, Jr., secretary, Box 31, Allenton, R. I.

SASKATCHEWAN—June 17, 1958, University of Saskatchewan, Saskatoon, Sask. Fred M. Clark, secretary, 619 9th Ave., Saskatoon, Sask.

SOUTH CAROLINA—June 19-20, 1958 (tentative), Columbia. H. L. Sutherland, secretary, P. O. Box 87, Union, S. Car.

SOUTH DAKOTA—June 23-24, 1958, Veterinary South Dakota State College, Science Department, Brookings. Dr. M. D. Mitchell, secretary, Livestock Sanitary Board, Pierre, S. Dak.

TENNESSEE—June 23-24, 1958, Nashville, Tenn. W. O. Greene, secretary, 4119 Hillsboro Road, Nashville, Tenn.

TEXAS—May 26-28, 1958, Texas A & M College, College Station, Texas. T. D. Weaver, executive secretary, Texas State Board Veterinary Medical Examiners, 207 Capital National Bank Bldg., Austin 16, Texas.

UTAH—June 19-20, 1958, State Capitol Building, Salt Lake City, Utah. Obtain Application from Mr. Frank Lees, State Capitol Bldg., Salt Lake City. Dr. Wayne Binns, Chairman, Utah State Veterinary Board of Examiners, Utah State University, Logan, Utah.

VIRGINIA—June 26, 1958, Capitol Building, Richmond, Va. T. N. Burton, secretary, Department of Professional and Occupational Registration, P. O. Box 1-X, Richmond, Va.

WASHINGTON—May 30-June 1, 1958, Pullman, Wash. Thomas A. Carter, Administrator, Professional Division, State Department of Licenses, Olympia, Wash.

DEATHS

Star indicates member of AVMA

★**Forrest R. Cameron** (MCK '18), 63, Hawley, Minn., died Jan. 12, 1958. Dr. Cameron had served with the ARS, U.S. Department of Agriculture. He was admitted to the AVMA in 1952.

W. C. Carnes (MCK '14), 68, Shelbyville, Ill., died Feb. 22, 1958. Dr. Carnes had served Shelbyville as mayor as well as veterinarian. He is survived by three daughters.

Harold J. Jewell (KSC '35), 47, Sulphur, Okla., died March 8, 1958. Dr. Jewell had served in World War II. He had been a member of the AVMA.

John E. Marston (CVC '17), 87, Manchester, N. H., died Feb. 13, 1958. Dr. Marston had practiced for 40 years and then, for 25 years, had raised and trained harness horses. His widow and two sons survive.

Otto R. Marzillier (CVC '18), 76, Jefferson, Wis., died Feb. 20, 1958. Dr. Marzillier had practiced in Jefferson for 40 years. He is survived by a son.

G. A. Meyer, 75, Pine Lake, Wis., died Feb. 17, 1958. Dr. Meyer had served in the Veterinary Corps, U. S. Army, during World War I. Since his retirement he had made his home in Appleton, Fla., and in Pine Lake. His widow, a daughter, and four sons survive.

William C. Muirhead (WES '06), 87, Bradshaw, Neb., died Feb. 17, 1958. Dr. Muirhead is survived by his widow, two daughters, one son, and eight grandchildren.

★**Clarence R. Palmer** (KCV '16), 75, Palo Alto, Calif., died Feb. 17, 1958. Until he retired five years ago, Dr. Palmer had served as veterinarian with the California Department of Agriculture for 16 years. Prior to that, he had served in the same capacity in the U.S. Department of Agriculture. He was made a life member of the AVMA in 1956. Dr. Palmer is survived by his widow, a daughter, two sons, and three grandchildren.

Leon L. Parker (COR '10), 72, Catskill, N. Y., died Dec. 14, 1957, of a heart ailment. Dr. Parker is survived by his widow and two granddaughters.

J. A. Price, 67, Paducah, Ky., died Feb. 10, 1958. Dr. Price had served McCracken County as veterinarian and livestock inspector for six years. He was a veteran of World War I. His widow and a daughter survive.

Arthur F. Pynn (CVC '14), 64, Oconomowoc, Wis., died Feb. 21, 1958. Dr. Pynn had served with the Wisconsin Department of Agriculture for 39 years. He retired in June, 1957, due to ill health.

★**Wilfred H. Timmons** (CIN '09), Monticello, Ind., died Jan. 31, 1958. Dr. Timmons was admitted to the AVMA in 1913 and made a life member in 1953. He is survived by his widow.

Harry C. Ward (CVC '10), 73, Fulton, Mo., died Feb. 25, 1958. Dr. Ward had practiced in Fulton for 45 years. His widow survives.

Charles T. Windrath (ISC '17), 65, Sibley, Iowa, died Feb. 7, 1958. Dr. Windrath had served as poultry inspector for the U.S.D.A. He is survived by his widow, a son, a daughter, and two grandchildren.

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Other Deaths Reported.—The following deaths have been reported. The usual information for an obituary was not supplied.

Robert V. Fox (ONT '22), 62, Alba, Mo., died Feb. 1, 1958.

Evan O. Burgess (MCK), 70, Conneaut, Ohio, died Dec. 21, 1957.

William A. Martin, Seymour, Ind., died March 3, 1958.

PUBLISHED REPORTS OF EXPERIENCE WITH

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Reprint from the JOURNAL of the American Veterinary Medical Association, vol. 124, No. 922, Jan., 1954, pp. 19-20.

A Note on the Use of Surital Sodium Anesthesia in Swine

H. W. DUNNE, D.V.M., Ph.D., and Captain S. C. BENBROOK, V.C., U.S. Army

East Lansing, Michigan

THE NEED for a satisfactory anesthetic in swine practice has long been recognized by veterinary surgeons and research workers throughout the nation. Until recently, most of the anesthetics available seemed to be contraindicated in some phase of application. They proved to be either toxic, resulting in frequent deaths, or the duration of anesthesia was too long and incurred time-consuming attention following an operation. Floundering and struggling associated with recovery from anesthesia of

Use of Surital Sodium and Curare in Small-Animal Surgery

W. O. Brinker, D.V.M., M.S.

Michigan State College

East Lansing, Mich.

Reprint, The North American Veterinarian, Vol. 32, December, 1951.

Reprinted from VETERINARY MEDICINE, Vol. XLVII, No. 3, March 1952

Surital Sodium And Morphine Anesthe In Canine Surgery

W. C. YOUNG, JR.,* D.V.M. and F. E. EADS D.V.M., M.S., Detroit, Michi

THE use of surital** sodium (sodium 5-allyl-5-(1-methylbutyl)-2-thiobarbiturate), a recently developed ultra-short acting thiobarbiturate, has been reported by several workers. It could be called the thiopentalogue of seconal** and as pentothal** sodium could be considered the thiosecobarbiturate of nembutal**.

In most instances, intravenously. Howe Dillon and Dennett' administration to re in this manner has nary medicine.

In five experim

Surital Sodium Anesthesia in Canine Surgery

H. B. ROBERTS, D.V.M., W. E. WENDT, D.V.M., C. C. WAGNER, D.V.M., M.Sc., T. F. REUTNER, D.V.M., M.S.

Reprint from the JOURNAL of the American Veterinary Medical Association, Vol. CXVIII, No. 888, March, 1951, pp. 191-193.

Reprint from the JOURNAL of the American Veterinary Medical Association, vol. 122, No. 912, May, 1953.

Dosage Studies of Surital Sodium in Dogs

ROBERT F. BORGMAN D.V.M., M.S.

Asheville, North Carolina

RECENTLY, a new thiobarbiturate, surital sodium, has been offered for limited clinical trial. Clinical reports have shown surital sodium to be an excellent anesthetic for operations of short duration in dogs. Its anesthesia is smooth and relatively free from excitement and irritability. When injected intravenously at hourly intervals,

RESULTS

A total of 11 dogs were used. The average dose necessary for a 7 min. surgical anesthesia was 27 mg. of surital sodium per kg. body weight. Of the 2 dogs an

TABLE I.—Results of Surital So

Reprinted from VETERINARY MEDICINE, Vol. L, No. 8, p. 265, August 1

Intrathoracic Anesthesia In Cats

MARK STERNFELS, D.V.M., Mount Vernon, New York

WHEN a human patient is suffering from an ailment which calls for surgical interference, laboratory techniques are employed to aid in the diagnosis. Preoperative injections of dextrose and saline solutions are given to raise blood sugar levels for prevention of shock and dehydration.

The anesthetist is usually part of the surgical

Surgery performed under anesth by intrathoracic administration cluded: Teeth cleaning and ex trimming and removal of injure abscesses, removal of cysts, pr tomies, castration, cesarea treatment, castration, accident cases, r fracture, and for x-ray diagno

Surital Sodium, A New Anesthetic and Hypnotic

Studies in Dogs

T. F. REUTNER, D.V.M., M.S., and O. M. GRUHZIT, M.S., M.D.

Detroit, Michigan

Reprint from the Journal of the American Veterinary Medical Association, Vol. CXIII, No. 908, October, 1948, pp. 207-208.

Surital Sodium Anesthesia in Dogs and Cats A Symposium Covering 7522 Patients

F. E. EADS, D.V.M., M.S., *Detroit, Mich.*

Although intravenous anesthesia is one of the oldest means of rendering patients insensible to pain, the method has been actively studied only within the past 15 years. So far as is known, the earliest attempt to produce anesthesia by vein was made in 1665 when a solution of opium was given by means of a

covery. They estimate that the approximately 1.5 times that of the dog. Similar results were Kelly, et al.** Surital seems slowly in dogs than does pen there appears to be less salura inactivation mechanisms wher

(*Reprinted from* THE CALIFORNIA VETERINARIAN, Sept.-Oct., 1953.)

These and other publications on Surital available on request

Department of Veterinary Medicine

PARKE, DAVIS & COMPANY

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MEDICAMENTA VERA

Skyline of New York City, with United Nations Buildings on the Left

Postconvention Tours Offered to New York and Bermuda

Ninety-Fifth Annual Meeting—Philadelphia August 18-21

AVMA members, their wives, and others who attend the Philadelphia Convention will have an opportunity for well-planned and inviting relaxation, sight-seeing in New York City, and a vacation trip to Bermuda through arrangements completed with Happiness Travel Service of Chicago.

An illustrated folder about the travel plans will be mailed later this month to all AVMA members. Other veterinarians and their wives are invited to join the tours and may obtain a folder by writing to the Association's headquarters office.

Choice of Three-day and Four-Day Weekends in New York

Participants will have a choice of three days and two nights in New York City or four days and three nights. For those who take the Bermuda trip, there will be only one "night at the Waldorf." In all cases, participants will leave Philadelphia by rail on Thursday, August 21, arriving in New York in the late afternoon. Hotel accommodations will be at the glamorous Waldorf-Astoria and the holiday starts with a cocktail party and dinner there on Thursday night.

New York Holiday

Friday and Saturday (August 22-23), or Friday, Saturday, and Sunday (August 22-24), as the case may be, will be devoted to sight-seeing, a cruise by boat around Manhattan Island, with visits to famous night clubs and restaurants and a Broadway show in the evenings.

Bermuda Holiday

Those who select this trip will leave New York by air on Friday morning, August 22, for the three-hour flight to Bermuda. There, accommodations will be provided at the Castle Harbor Hotel, the

Aerial View of Hamilton, Capital City of Bermuda

Castle Harbor Hotel

most modern and luxurious on the islands; it is a completely self-contained resort with its own pool, private beach, boat dock, tennis courts, and tropical gardens; guests may play golf at the Castle Harbor links or at neighboring courses such as St. George's and Mid-Ocean.

The return from Bermuda will be on Saturday, August 30.

Costs of Tours

The per-person cost of the various itineraries is as follows:

1) New York Weekend —
3 days-2 nights at the Waldorf ..... $ 82.50

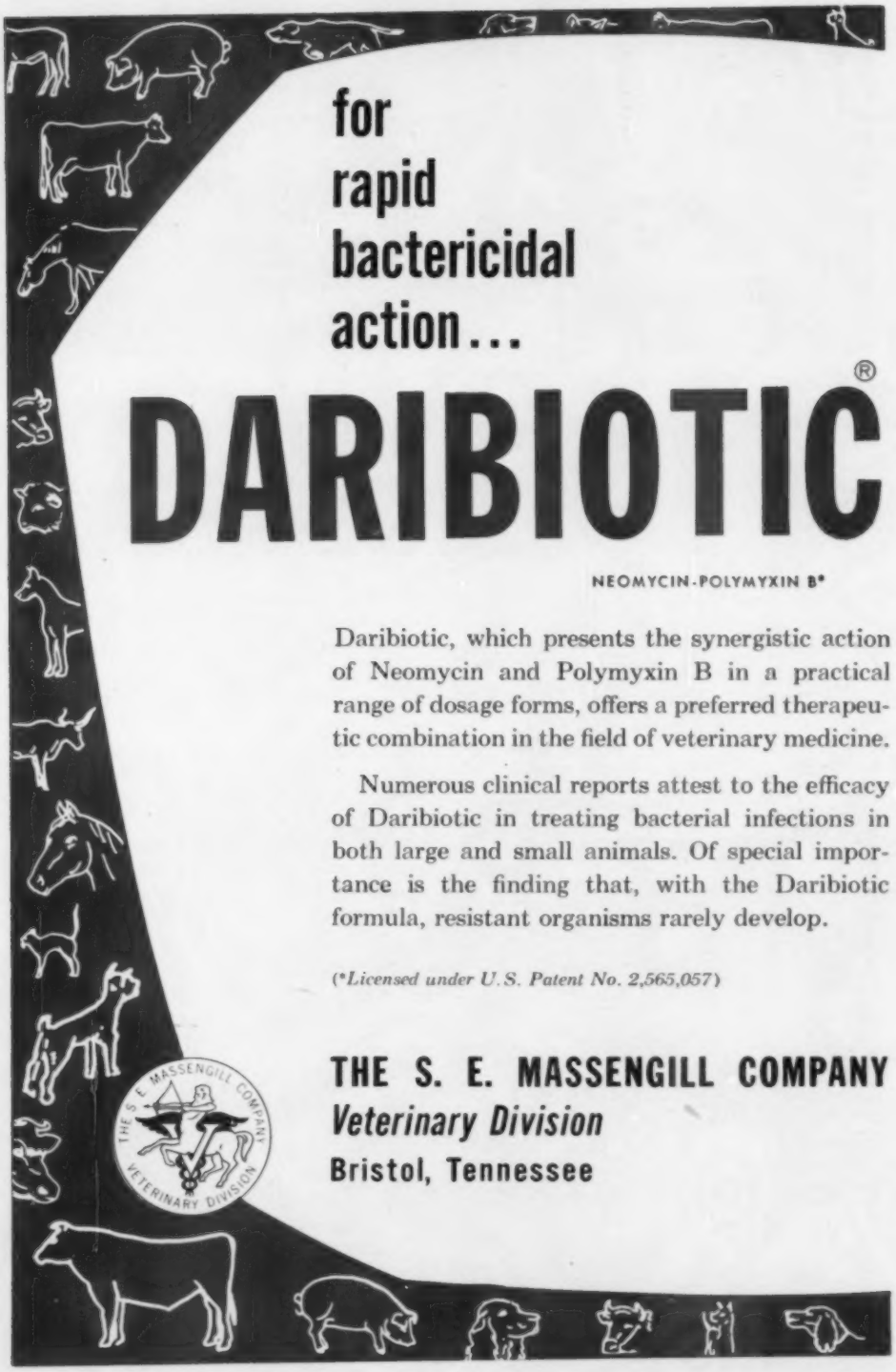
2) New York Weekend —
4 days-3 nights at the Waldorf $122.00

3) Bermuda Holiday $357.00

These costs include rail transportation, Philadelphia to New York, accommodations and certain meals at the Waldorf, sightseeing as specified, special features, baggage handling, and escort service on the Bermuda trip.

Complete information and details of what is and what is not included will be given in the tour leaflet which will be mailed later.

A Carriage Ride in Somerset, Bermuda



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Daribiotic, which presents the synergistic action of Neomycin and Polymyxin B in a practical range of dosage forms, offers a preferred therapeutic combination in the field of veterinary medicine.

Numerous clinical reports attest to the efficacy of Daribiotic in treating bacterial infections in both large and small animals. Of special importance is the finding that, with the Daribiotic formula, resistant organisms rarely develop.

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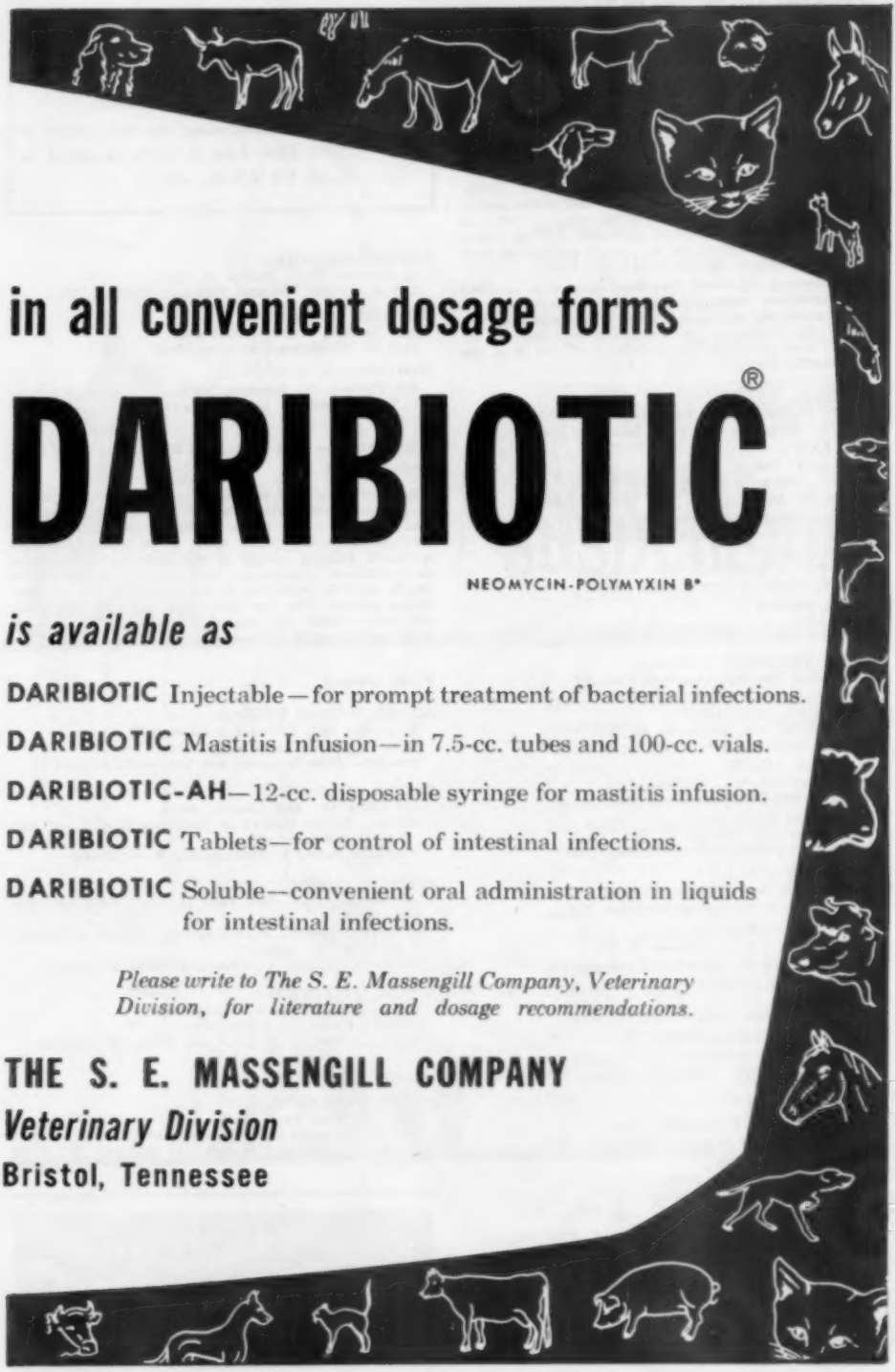

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APPLICATIONS

Applicants—Members of Constituent Associations

In accordance with paragraph (e) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., the names of applicants residing within the jurisdictional limits of the constituent associations shall be published once in the JOURNAL.

The following applicants have been certified as members of the constituent association that has jurisdiction over the area in which the applicant resides. This certification was made by the secretary of the constituent association in accordance with paragraph (c) Section 2, Article X of the Administrative Bylaws.

BAGDONAS, VALENTINE,
519 E. Hudson Street, Wellsville, Mo.,
D.V.M., Veterinary College of Hannover, 1946.

BAUS, R. W.,
Ipswich, S. Dak.,
D.V.M., University of Minnesota, 1956.

BERGERON, PIERRE,
223 Bois Franc, Saint-Laurent, Que.,
V.M.D., Ecole de Medicine Veterinarie, 1953.

FIELDER, JOE L.,
Belt Blvd. & Midlothian Pike, Richmond 25, Va.,
D.V.M., Michigan State College, 1949.

FOSTER, HENRY L.,
82 Brookline St., Chestnut Hill Newton 67, Mass.,
D.V.M., Middlesex Veterinary College, 1946.

HARRIS, THEODORE WILLIAM,
2434 Green Bay Rd., Highland Park, Ill.,
D.V.M., Middlesex University, 1943.

HENNINGER, WILLIAM F.,
2011 Harman Ave., Baltimore 30, Md.,
D.V.M., Royal Veterinary College, 1914.

KALANTAR, LEVON,
130 E. Alameda Ave., Burbank, Calif.,
D.V.M., Middlesex University, 1944.

LEE, DENZIL B.,
9359 Vanderpoel, Chicago 20, Ill.,
V.M.D., University of Pennsylvania, 1956.

LEPON, EDWARD L.,
9524 Humble Rd., Houston, Texas,
D.V.M., A. & M. College of Texas, 1942.

McDONALD, ROBERT L.,
4570 Richmond Road, Bellaire, Texas,
D.V.M., A. & M. College of Texas, 1951.

MASTERSON, KENNETH G.,
Route 1, Ault, Colo.,
D.V.M., Colorado State University, 1955.

MEARS, REX HAWTHORNE,
P. O. Box 999, Salmon Arm, B.C.,
M.R.C.V.S., Royal Veterinary College, University of London, 1934.

MOREAU, PIERRE,
19 Tourigny St., Victoriaville, Que.
D.V.M., Ecole de Medecine-Veterinaire, 1950.

NUTTER, ROBERT M.,
705 Main St., Hanson, Mass.,
D.V.M., Middlesex University, 1942.

PLATT, HAROLD,
3130 E. Livingston Ave., Columbus 13, Ohio,
D.V.M., Middlesex University, 1947.

SLAY, CLYDE, Jr.,
4570 Richmond Road, Bellaire, Texas,
D.V.M., A & M College of Texas, 1951.

TABLEMAN, HARVEY G.,
Box 81, Crystal Lake, Ill.,
D.V.M., Middlesex University, 1944.

Gaines Award — Correction

Deadline for nominations for this award is *June 1, 1958* (not June 1, 1957, as stated in April JOURNAL, adv. p. 31).

WEINER, BENJAMIN,
43 Newman Road, Malden 48, Mass.,
D.V.M., Alfort National Veterinary College, 1951.

WIGNALL, ROBERT F.,
1500 Gorham St., Lowell, Mass.,
D.V.M., Middlesex University, 1943.

WHITESTONE, BURTON D.,
820 Pleasant St., Belmont, Mass.,
D.V.M., Middlesex University, 1942.

Applicants — Not Members of Constituent Associations

In accordance with paragraph (e) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., notice of all applications from applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months. The first notice shall give the applicant's full name, school, and year of graduation, post office address, and names of his endorsers.

First Listing

ADAMS, EUGENE WILLIAM,
P. O. Box 267, Tuskegee Institute, Ala.
D.V.M., Kansas State College, 1944.
Vouchers: John B. Smith and Charles G. Rickard.

BAJWA, GURWANT S.,
210 Cedar St., East Lansing, Mich.
B.V.Sc., Punjab College of Veterinary Science and Animal Husbandry, 1951.
Vouchers: Robert F. Langham and R. D. Barner.

EISCHEN, JOHN NICHOLAS,
1st Hospital Unit, 45th Field Hospital, APO 19, New York, N.Y.,
D.V.M., Oklahoma State University, School of Veterinary Medicine, 1956.
Vouchers: William L. Hays and Elmer W. Young.

MILLER, EVERETT B.,
Poortlaan 2, Wassenaar, Netherlands,
V.M.D., University of Pennsylvania, 1940.
Vouchers: Herbert F. Sibert and Robert K. Dunton.

Second Listing

SMITH, RICHARD E.
APO 132, New York, N. Y.
D.V.M., Colorado State University, 1952.
Vouchers: William J. Welch and Rowland W. Rushmore.

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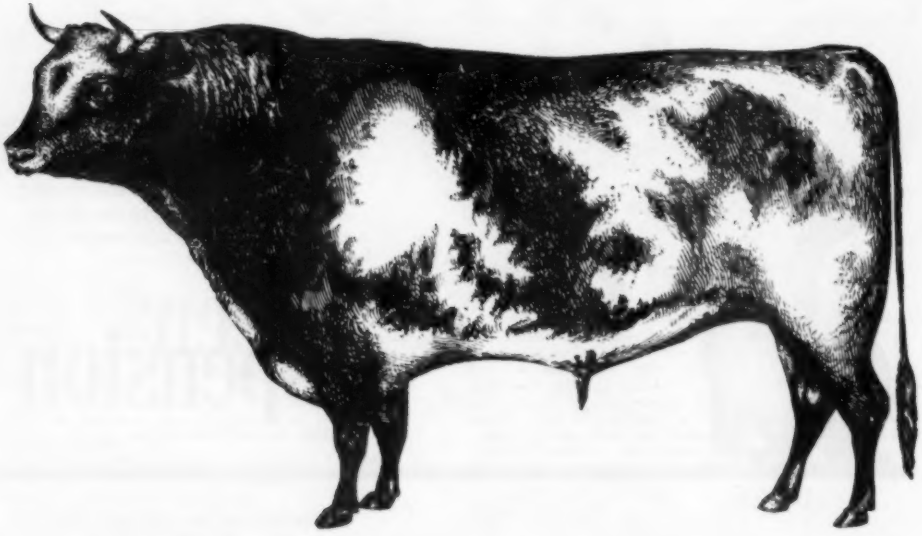

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ADDITIONAL INFORMATION: Write to Veterinary Medical Division, CIBA, Summit, New Jersey, requesting Veterinary Therapeutic Index.

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COMING MEETINGS

Oklahoma State University. Oklahoma conference for Veterinarians. College of Veterinary Medicine, Oklahoma State University, Stillwater, May 5-6, 1958. Lester Johnson, Department of Veterinary Medicine and Surgery, chairman.

Eastern Iowa Veterinary Association. Twenty-fourth annual all-day Practitioners' clinic, Hawkeye Downs, Cedar Rapids, Iowa. May 6, 1958. F. E. Brutsman, Traer, Iowa, secretary.

District of Columbia Veterinary Medical Association. Third quarterly meeting. Armed Forces Institute of Pathology, Walter Reed Army Medical Center, Washington 25, D.C., May 13, 1958. W. I. Gay, 5200 Chandler St., Bethesda, Md., secretary-treasurer.

Michiana Veterinary Medical Association. Bi-annual clinic. Goshen Sales Barn, Goshen, Ind., May 14, 1958, at 1:00 p.m. Dr. S. Williamson, 217 W. Chippewa, South Bend, Ind., chairman.

Woodbury County Medical Society and Interstate Veterinary Medical Association. Leptospirosis symposium. Hotel Mayfair, Sioux City, Iowa, May 15, 1958.

Florida Conference for Veterinarians. First annual conference. Health Center Auditorium, University of Florida, Gainesville, May 17-18, 1958. Dr. M. W. Emmel, general conference chairman.

Michigan Veterinary Medical Association. Annual meeting. Grand Hotel, Mackinaw Island, June 2-4, 1958. Charles Coy, Hillsdale, general chairman.

Texas A. & M. College. Eleventh annual Texas conference for veterinarians. School of Veterinary Medicine, Texas A. & M. College, College Station, June 5-6, 1958. R. D. Turk, chairman.

Kansas State College. Annual conference for veterinarians. School of Veterinary Medicine, Kansas State College, Manhattan, June 5-7, 1958. E. E. Leasure, dean.

Montana Veterinary Medical Association. Summer meeting. Florence Hotel, Missoula, June 16-18, 1958. G. A. Morrison, 316 Central Ave., Great Falls, secretary.

Georgia-South Carolina Veterinary Medical Association. Joint Meeting, Bon Air Hotel, Augusta, Ga., June 19-21 1958. A.M. Mills, 325 Pinecrest Drive, Athens, Ga., secretary, program committee.

Idaho and Wyoming Veterinary Medical Association. Joint meeting. Wort Hotel, Jackson Hole, Wyo., June 21-23, 1958. A. P. Schneider, 3025 N. 23rd St., Boise, Idaho, IVMA secretary; J. F. Ryff, P.O. Box 960, Laramie, Wyo., WVMA secretary.

California Veterinary Medical Association. Seventieth annual meeting. Hotel St. Claire, San Jose, June 23-25, 1958. C. H. Ozanian, 10326 Artesia Blvd., Bellflower, program chairman; Chas. S. Travers, 3004 16th St., San Francisco, executive secretary.

Maritime Veterinary Associations. Joint conference. Mount Allison University, Sackville, N.B., June 24-26, 1958. Dr. J. F. Frank, Box 310, Sackville, N.B., chairman.

North Carolina State Veterinary Medical Association. Fifty-seventh annual meeting. The Washington Duke Hotel, Durham, June 24-26, 1958. C. J. Lange, 3741 Hi-Point Rd., Greenboro, secretary-treasurer.

Utah Veterinary Medical Association. Annual meeting. Ogden, June 25-26, 1958. J. A. Thomas, P.O. Box 592, Provo, secretary.

Alabama Polytechnic Institute. Annual conference for veterinarians. School of Veterinary Medicine, Auburn, July 20-23, 1958. T. C. Fitzgerald, program chairman.

NEW BOOK

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Pathologist, Angel Memorial Animal Hospital, Boston, formerly Chief, Veterinary Pathology Section, Armed Forces Institute of Pathology, Washington, D.C.

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Canadian Veterinary Medical Association. Tenth annual Convention. Royal Alexandra Hotel, Winnipeg, Manitoba. July 21-23, 1958. Claude Kealey, 1195 Wellington St., Ottawa 3, Ont., executive secretary.

New York State Veterinary Medical Society. Sixty-seventh annual meeting. Concord Hotel, Kiamesha Lake, N. Y., Sept. 4-6, 1958. Miss Joan S. Halat, 803 Varick St., Utica, N. Y., executive secretary.

Washington State Veterinary Medical Association. Annual meeting, Olympia, Sept. 8-9, 1958, Fred M. Shigley, program chairman. Robert M. Ford, 2406 Boyer Ave., Seattle, executive secretary.

New England Veterinary Medical Association. Annual meeting. Hotel Wentworth, Portsmouth, N. H., Sept. 21-24, 1958. C. Lawrence Blakely, 100 Longwood Ave., Boston 15, Mass., secretary-treasurer.

District of Columbia Veterinary Medical Association. Annual all-day meeting. Walter Reed Army Medical Center, Washington, D.C., Oct. 14, 1958. W. I. Gay, 5200 Chandler St., Bethesda, Md., secretary-treasurer.

Eastern Iowa Veterinary Association. Forty-fifth annual meeting. Hotel Roosevelt, Cedar Rapids, Iowa. Oct. 16-17, 1958. F. E. Brutsman, Traer, Iowa, secretary.

Texas Veterinary Medical Association. Annual meeting. Stephen F. Austin Hotel, Austin, Oct. 19-21, 1958. Paul B. Blunt, 710 Maverick Bldg., San Antonio, secretary.

Southern Veterinary Medical Association. Annual meeting. Claridge Hotel, Memphis, Tenn., Oct. 26-30, 1958. A. A. Husman, P.O. Box 91, Raleigh, N. Car., secretary.

Oklahoma Veterinary Medical Association. Annual meeting. Mayo Hotel, Tulsa, Jan. 25-27, 1959. M. N. Riemenschneider, 122 State Capitol Bldg., Oklahoma City, secretary.

Foreign Meetings

British Caribbean Veterinary Association. Third veterinary convention. Trinidad, B.W.I., May 25-31, 1958. S. P. Bennett, c/o Dept. of Agric., St. Clair, Port of Spain Trinidad, B.W.I., president.

Sixth International Congresses on Tropical Medicine and Malaria. Lisbon, Portugal, Sept. 5-13, 1958. Professor Manuel R. Pinto, Institute of Tropical Medicine, Lisbon, secretary-general. (Membership application forms may be obtained by U.S. veterinarians by writing to the AVMA.)

German Veterinary Association. Regular biennial meeting. Hannover, Germany, Sept. 19-21, 1958. Dr. Karl Ohly, 123 Forsthausstrasse, Frankfurt/Main, Germany, president.

Third World Congress on Fertility and Sterility. Amsterdam, Holland, June 7-13, 1959. Dr. L. I. Swaab, Sint Agnietenstraat 4, Amsterdam, Holland, honorary secretary.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. Dr. G. W. Jones, Main St., Prattville, Ala., secretary-treasurer.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

North Alabama Veterinary Medical Association, the second Thursday of November, January, March, May, July, and September, in Decatur, Ala. Ray A. Ashwander, Decatur, Ala., secretary.

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North East Alabama Veterinary Medical Association, the second Tuesday of every other month. Leonard J. Hill, P.O. Box 761, Gadsden, Ala., secretary-treasurer.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2 Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December. Herb Warren, 3004 16 St., San Francisco, Calif., executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643-N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. R. M. Granfield, 2600 W. El Camino Real, San Mateo, Calif., secretary-treasurer.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert L. Chandler, P.O. Box 8, Ukiah, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Chapter SCVMA, the second Tuesday of each month at 7:30 p.m., Hody's Restaurant, North Hollywood, Calif. Dr. V. H. Austin, 14931 Oxnard St., Van Nuys, secretary-treasurer.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. Dr. Rolf Reese, 23815 Ventura Blvd., Calabasas, Calif., secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinary Medical Association, the second Thursday of each month. D. E. Britten, 544 N. Ben Maddox, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth

(Continued on adv. p. 42)

The total world-catch of fish continues to increase. It now approaches 30 million metric tons per year.—*Sci. News Letter (Feb. 8, 1958)*: 94.

Pitman-Moore Announce New Officers

Pitman-Moore's president, Mr. Kenneth Valentine, announced recently the appointment of four new vice-presidents: Stewart R. Ruch, Dr. S. R. Bozeman, Dr. C. A. Bunde, and Harry Sefton.

Allen B. Faux is the new treasurer and John R. Jewett was named assistant secretary.

John R. Jewett

Mr. Jewett became associated with Pitman-Moore Company in 1948 as director of market research. In 1951, he became assistant advertising manager and, in 1953, he was named assistant to the executive vice-president. Mr. Jewett is a graduate of DePauw University.

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Available as orange scored tablets of 10, 50 and 100 mg.; and Oral Suspension, bottle of 60 cc.

REFERENCES: 1. Mosier, J. E.: Vet. M. 50:605 (Nov.) 1955. 2. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 3. Pollock, S.: J. Am. Vet. M. Ass. 129:274 (Sept.) 1956.

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Make your diagnosis from the picture below—then turn the page ▶

Figure 1

History.—A Thoroughbred stallion, 4 years old, with a history of chronic lameness in the left foreleg, was examined. He was less lame than he had been formerly. The left forefoot was smaller than the right. Radiographs were taken (lateral view) of both digits. The radiograph of the left digit is shown (fig. 1).

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—Chronic laminitis of the left forefoot of a horse.

Comment.—Notice the altered position of the third phalanx within the hoof (fig. 2) as compared to the normal foot (fig. 3).

Fig. 2—The abnormal corium (arrows) appears as a darkened line. Notice the concave dorsal line of the wall of the hoof, the increasing thickness of the wall as it develops distally, and the thin sole under the toe of the third phalanx.

As if rotated on a transverse axis, the toe of the bone is lowered into the sole and drawn away from the dorsal wall of the hoof. The chronic alterations in the laminar corium (see dark line) result in interference in the circulation to the hoof. In extreme cases, the dorsal wall of the hoof curves forward at it grows, and the sole is forced away from the wall, thus exposing the toe of the third phalanx.

Fig. 3—Lateral radiograph of a normal foot. Notice the more uniform thickness of the wall and sole; also the straight dorsal line of the wall.

This case was submitted by Dr. F. J. Milne, M.R.C.V.S., professor of surgery and obstetrics, Ontario Veterinary College, Guelph.

Our readers are invited to submit histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

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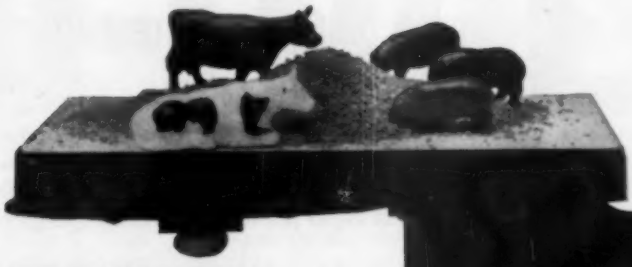
1. Vet. Med., 52:601-605
2. Brit. J. Nutr., 11:70-79
3. Biochem. & Physiol. Nutr., 2:439-440
4. Armour Research Foundation Report, Project No. C616
5. Ibid.
6. J. Dairy Sci., 40 (9):1087-1092



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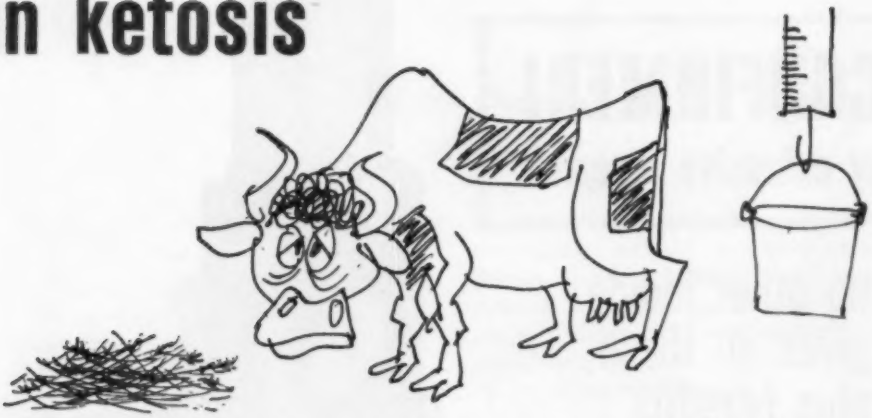

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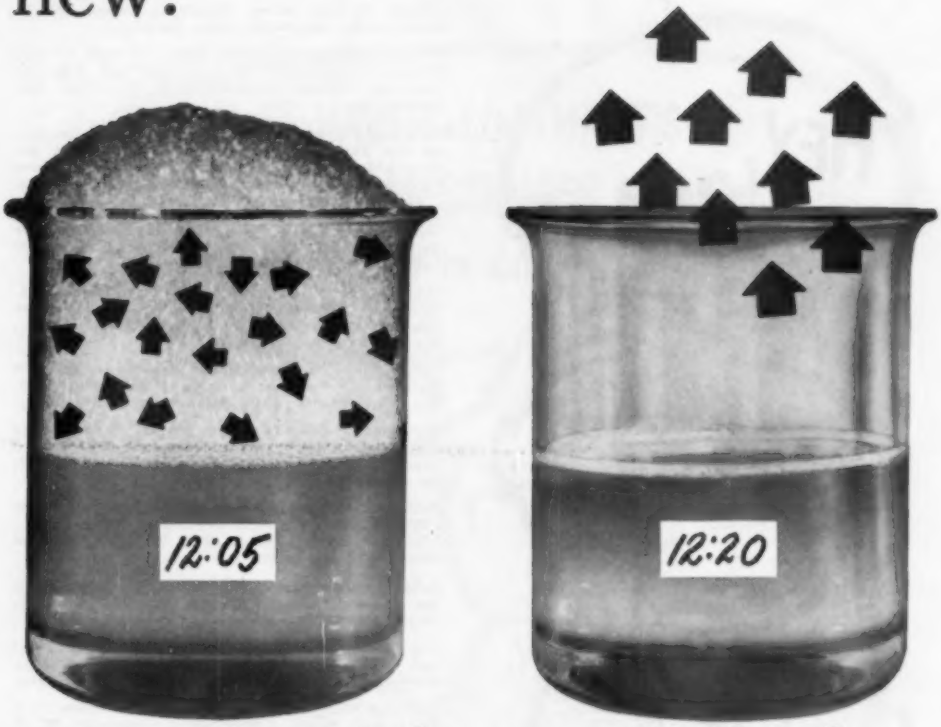

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Dosage—Cattle: intrarumenally or orally, 20-30 cc. Sheep and goats: 5-10 cc. Dose may be repeated in one hour, if necessary.

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Department of Veterinary Medicine, PFIZER LABORATORIES, *Division, Chas. Pfizer & Co., Inc.*, Brooklyn 6, N. Y.

58-4

Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyea, 5925 Edgewater Drive, Orlando, Fla., secretary.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, St. Petersburg. William F. Casler, 2540 30th Ave., N., St. Petersburg, secretary-treasurer.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Gecl, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4148 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla., secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the third Thursday of each month at the Elk's Home, 726 Peachtree St., Atlanta. Donald C. Ford, Forest Park, secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Charles H. Armstrong, 1021 Davis St., Evanston, secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. Alfred G. Schiller, Veterinary Clinic, University of Illinois, Urbana, secretary-treasurer.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P. O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August,

Right again—besides publishing pretty fair jokes (not to mention timely articles), the Haver-Lockhart Messenger is the most widely-used price bible for veterinarians. Published every two months, it's always up to date; always reflects the current market.

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58-4

at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Association, the second Thursday of each month at 6:30 p.m., usually in Cedar Rapids, Iowa. Dr. J. G. Irwin, Iowa City, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Lakes Veterinary Association, the first Tuesday of each month, September through May, at 6:30 p.m., at the Gardson Hotel, Estherville, Iowa. Barry Barnes, Milford, secretary.

North Central Iowa Veterinary Medical Association, the third Thursday of April, at the Warden Hotel, Fort Dodge, Iowa. H. Engelbrecht, P. O. Box 797, Fort Dodge, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Winneslick Hotel, Decorah, Iowa. 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northwest Iowa Veterinary Medical Association, the second Tuesday of February, May, September, and December, at the Community Bldg., Sheldon. W. Ver Meer, Hull, secretary.

Southeastern Iowa Veterinary Association, the first Tuesday of each month at Mt. Pleasant, Iowa. Warren Kilpatrick, Mediapolis, secretary.

Southwestern Iowa Veterinary Medical Association, the first Tuesday of April and October, Hotel Chieftain, Council Bluffs, Iowa. J. P. Stream, Creston, secretary.

Upper Iowa Veterinary Medical Association, the third Tuesday of each month at 7:00 p.m., at All Vets Center, Clear Lake, Iowa. Richard Baum, Osage, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Versailles Rd., Frankfort, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m., at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Norman Herbert, 3506 Joann Drive, Baltimore 7, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5054 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and

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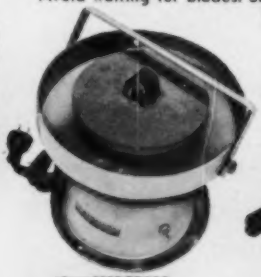

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*Jones, S. V.; Belloff, G. B., and Roberts, H. D. B.: Vet. Med. 51:413 (Sept.) 1956.

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August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Chester R. Pleggé, 4249 Peck St., St. Louis 7, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Thursday of each month at the Hotel President, Kansas City, Mo. Frank A. O'Donnell, Parksville, Mo., secretary-treasurer.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Elks Club, Hackensack. Burritt Lupton, 569 Franklin Ave., Wyckoff, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. R. M. Sauer, secretary.

NEW MEXICO—Bernalillo County Veterinary Practitioners Association, third Wednesday of each month, Fez Club, Albuquerque, N.M. Jack Ambrose, 3018 N. Rio Grande Blvd., Albuquerque, secretary-treasurer.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College, Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly, Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. T. L. James, Box 243, Newton, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Friday of each month at Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. J. E. Currie, 690 N. Leak St., Southern Pines, N. Car., secretary.

Western North Carolina Veterinary Medical Association, the second Thursday of every month at 7:00 p.m. in the George Vanderbilt Hotel, Ashville, N. Car. Vilu Lind, 346 State St., Marion, N. Car., secretary.

OHIO—Cincinnati Veterinary Medical Association, the third Tuesday of every month at Shuller's Wigwam, 6210 Hamilton Ave., at North Bend Road. G. C. Lewis, Cincinnati, Ohio, secretary-treasurer.

Columbus Academy of Veterinary Medicine, every month, September through May. E. M. Simonson, Columbus, Ohio, secretary-treasurer.

Cuyahoga County Veterinary Medical Association, the

first Wednesday in September, October, December, February, March, April, and May, at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. F. A. Coy, Cleveland, Ohio, secretary.

Dayton Veterinary Medical Association, the third Tuesday of every month. O. W. Fallang, Dayton, secretary.

Killbuck Valley Veterinary Medical Association, the first Wednesday of alternate months beginning with February. D. J. Kern, Killbuck, Ohio, secretary-treasurer.

Mahoning County Veterinary Medical Association, the third Tuesday of each month, at 9:00 p. m., Youngstown Maennerchor Club, Youngstown, Ohio. Sam Segall, 2935 Glenwood Ave., Youngstown, secretary.

Miami Valley Veterinary Medical Association, the first Wednesday of December, March, June, and September. J. M. Westfall, Greenville, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month except during the summer. R. W. McClung, Tiffin, Ohio, secretary-treasurer.

Northwestern Ohio Vererinary Medical Association, the last Wednesday of March and July. C. S. Alvanos, Toledo, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBrides Emerald Lounge. Canton, Ohio. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

Summit County Veterinary Medical Association, the last Tuesday of every month (except June, July, and August), at the Mayflower Hotel, Akron, Ohio. M. L. Scott, Akron, Ohio, secretary-treasurer.

Tri-County Veterinary Medical Association, the fourth Wednesday of January, May, and September. Mrs. R. Slusher, Mason, Ohio, secretary-treasurer.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 530 S. Madison St., Tulsa, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m., Ireland's Restaurant, Lloyds', 718 N.E. 12th Ave., Portland. Donald L. Moyer, 8415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

Willamette Veterinarian Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Marvin M. Corff, McMinnville, Ore., secretary

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania, School of Veterinary Medicine. Raymond C. Snyder, N. E. Corner 47th St. and Hazel Ave., Philadelphia 43, Pa., secretary.

Lehigh Valley Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Pennsylvania Northern Tier Veterinary Medical Association, the third Wednesday of each odd numbered month. R. L. Michel, Troy, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S Car., secretary.

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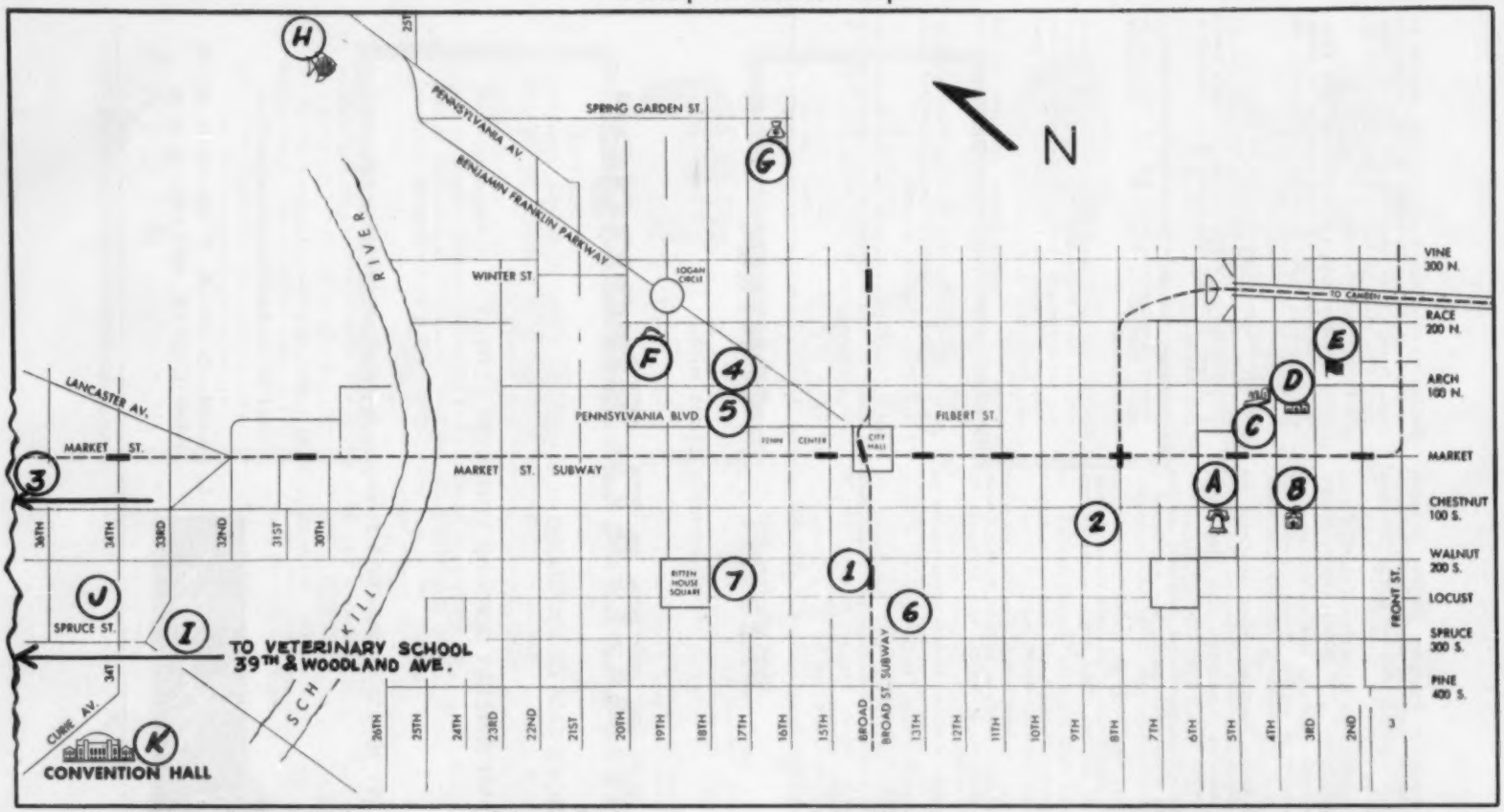

1. Bellevue-Stratford Hotel
2. Benjamin Franklin Hotel
3. Penn-Sherwood
4. Robert Morris (not pictured)
5. Sheraton Hotel
6. Sylvania Hotel
7. Warwick Hotel

A. Independence Hall
B. Carpenters' Hall
C. Franklin's Grave
D. Friends' Meeting House
E. Betsy Ross House
F. Academy of Natural Sciences
G. U.S. Mint (Guided Tours)
H. Aquarium
I. University Museum
J. University of Pennsylvania
K. Convention Hall

HOTEL RESERVATIONS — PHILADELPHIA CONVENTION

Ninety-Fifth Annual AVMA Meeting, Aug. 18-21, 1958

All requests for hotel accommodations will be handled by a Housing Bureau in cooperation with the Committee on Local Arrangements. The Bureau will clear all requests and confirm reservations.

Hotels and Rate Schedule

Hotel	Single	Double	Twin	Suite
1. Bellevue-Stratford †	$ 9.00-11.00	$12.00-16.00	$12.00-17.00	$30.00-50.00*
2. Benjamin-Franklin †	9.00-11.00	12.00-15.00	16.00-18.00	.....
3. Penn Sherwood †	6.00- 7.50	11.00	13.50-14.00	15.50*
4. Robert Morris †	6.00	9.50	11.00	.....
5. Sheraton †	9.85-13.50	13.00	15.00-17.00	30.50-41.00*
6. Sylvania ††	7.50- 9.50	.....	10.00-13.00	25.00-27.50*
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†† Partly Air-Conditioned
* 2-room suite

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First choice hotel _____	_____ Double Room(s) @ $_____
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TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary Ct., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the Second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blackburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. I. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). R. J. O'Hern, P.O. Box 617, Cumberland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Parley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. E. S. Scobell, Rt. 2, Wausau, Wis., secretary.

To identify babies in a busy delivery room, a metal clamp with the initials of the doctor and the mother is placed, close to the body, on the umbilical cord, before it is severed. It usually drops off in a day. —*Sci. News Letter (Jan. 18, 1958): 38.*

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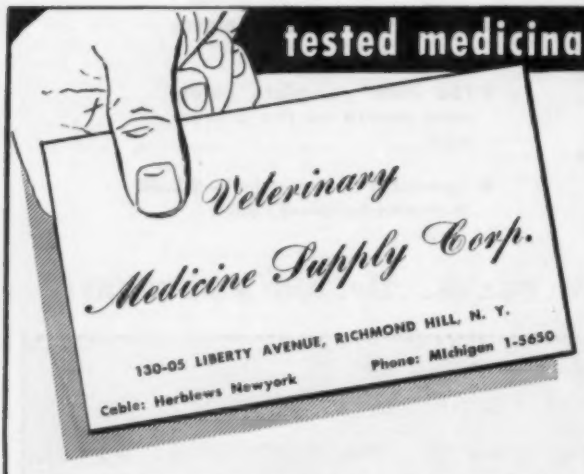

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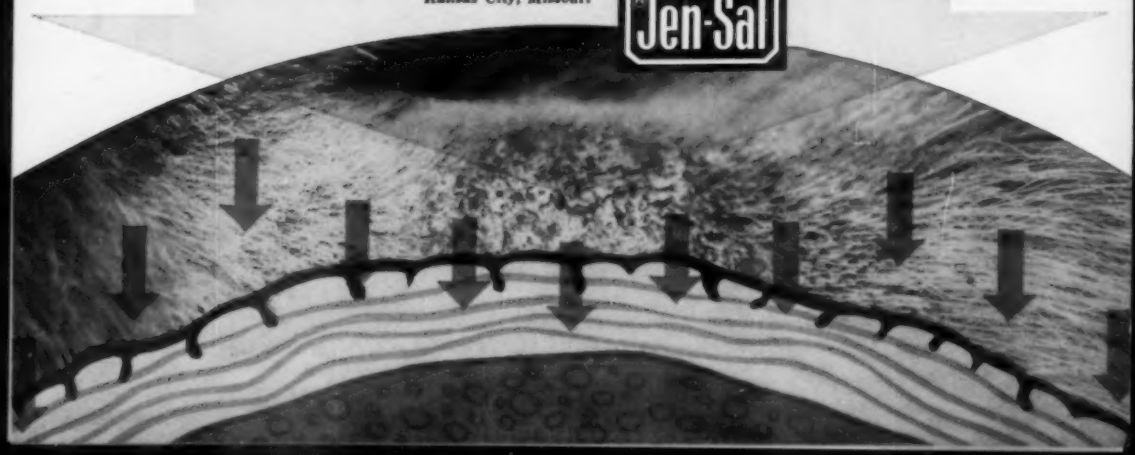
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